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The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Crops Research Division serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

13 FEB 1960

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ACCEPTANCE OF MANUSCRIPTS

The increase in the volume of pertinent material offered for publication in the Plant Disease Reporter has made it necessary to limit the subject matter and the length of articles accepted. The subject matter should emphasize new things in plant pathology, such as new records of disease occurrence, serious outbreaks and epidemics, conditions affecting development of plant diseases, techniques of investigation including instrumentation, new discoveries in control including new materials and their evaluation. Manuscripts will be limited to 12-double-spaced typed pages, including tables, graphs, and photographs. Because of reproduction costs photographs should be kept to a minimum. Insofar as possible, material should be presented as graphs rather than tables. Papers cannot be accepted for publication that report routine control experiments, reviews, bibliographies without annotation, results of routine surveys, mere summaries or lists of plant diseases. By following this procedure we hope to continue publishing all articles promptly.

Paul R. Miller

Manuscripts for and correspondence about this publication should be sent to:

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Mycology and Plant Disease Reporting Section
Crops Protection Research Branch
Plant Industry Station
Beltsville, Maryland

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OBSERVATIONS ON COTTON RUST (PUCCINIA STAKMANII) UNDER SEVERE DISEASE CONDITIONS[1]

T. E. Smith[2]

Abstract

Southwestern cotton rust (Puccinia stakmanii) [= P. cacabata Arth. & Holw.] was destructive in New Mexico in 1959. The more severe infestations were associated with a record total rainfall for July and August. Annual grama grasses (Bouteloua aristidoides and B. barbata) were the principal grass hosts. Moderate to heavy summer showers in 1957, 1958, and 1959 have built up the amount of susceptible grass and inoculum to such an extent that the rust potential is high for 1960.

Southwestern cotton rust (Puccinia stakmanii Presley & King) has occurred in New Mexico in negligible amounts for many years. In 1959 a severe outbreak of this disease occurred in the southwestern area of the State. In Dona Ana County, near Radium Springs, rust occurred in localized infestations. In Luna County rust was light to moderate in certain communities. In Hidalgo County rust was severe, causing 50 to 75 percent crop loss in the vicinity of Rodeo. The losses were caused by premature defoliation due to 50 to 100 aecia per leaf, locule dwarfing due to infection on the carpel walls and peduncle infection (Fig. 1) which contributed to breakage and loss of mature bolls.

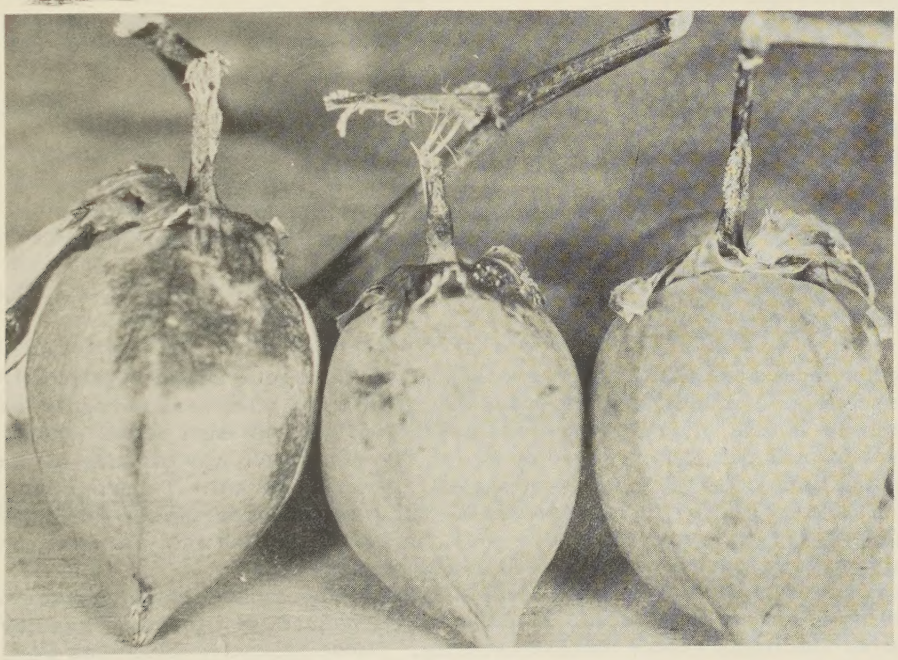

FIGURE 1. Pustules on the peduncles contributed to breakage and loss of mature cotton bolls.

This epiphytotic on cotton was associated with increased rainfall in two different ways. First, July and August showers were rather heavy for 1957 and 1958. This permitted the grass hosts to increase to an unusual degree throughout the region. Second, in 1959 the total rainfall for July and August was 8.23 inches, a record amount for these months during the 25 years of

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[1]Journal Series No. 138, Agricultural Experiment Station, New Mexico State University.

[2]Associate Plant Pathologist, Department of Botany and Entomology, Agricultural Experiment Station, New Mexico State University. The writer acknowledges J. J. Norris for identification of grasses, and J. J. Durkin and J. M. White for photographic assistance, and G. B. Hoff for certain collections of infected cotton.

complete records at the Rodeo cooperative weather station. This unusually heavy rainfall was favorable for extensive disease development as reported earlier (1 and others).

These conditions also induced severe rust on the grama grasses (Bouteloua sp.) demonstrated (4, 5) to be the alternate hosts. Such a development permitted reliable observations on the rust reaction of various grass species that grew adjacent to infected gramas. The results are summarized in Table 1 and show that the annual gramas (B. aristidoides and B. barbata) were the principal grass hosts.

Table 1. Reaction of grass species to P. stakmanii under conditions of severe natural infection.

Grass species[a]	Literature reports	Reaction
Aristida adscenscionis		-[b]
Bouteloua aristidoides	+[c] (4)+(5)	+
B. barbata	+ (4)+(5)	+
B. curtipendula	- (4)+(5)	-
B. gracilis	+ (5)	-
Chloris virgata		-
Eragrostis barrelieri		-
Echinochloa colonum		-
Hilaria mutica		-
Muhlenbergia arenacea	- (5)	-
M. porteri	- (5)	-
Panicum obtusum		-
Scleropogon brevifolius		-
Sporobolus contractus	- (5)	-
S. cryptandrus	- (5)	-
Tridens pulchellus		-

[a] Named in accordance with: Hitchcock, A. S. 1951. Manual of the Grasses of the United States, U.S. Dept. of Agr. Misc. Publ. 200, revised by Agnes Chase.

[b] - = not infected.

[c] + = infected.

Two other fungi were observed in association with the aecial stage on cotton. Alternaria sp. occurred on leaf pustules as a thin black overgrowth and often caused zonate leaf-spot symptoms in the lamina adjacent to the rust pustules as well as "shot holes" where the infected pustules had dropped out. As reported by Brown (2), defoliation was striking on plants where Alternaria and rust occurred on the same leaves. The rust parasite Tuberculina persicina reported from Arizona (1) was widely distributed, being collected during the fall months in all fields examined. It was characterized by grey, brown or purplish warty stromata occurring on aecial pustules on the carpel walls (Fig. 2) and peduncles.

The amount of inoculum currently overwintering on the annual grama grasses is such that rust capability is very high for 1960. Because of this hazard the literature was reviewed to find measures that might reduce disease severity. These palliative measures were evaluated against field observations in 1959.

Elimination of grasses from cultivated fields and their borders is mentioned, as well as avoidance of planting cotton adjacent to non-cultivated grass land (3, 4, 5). This approach would have reduced rust damage under conditions of light to moderate attack, because most of the infection on cotton under such conditions occurred near mesa areas or water sources. However, under conditions of severe rust development infection was abundant in the center of large fields approximately a half mile from non-cultivated land. Duffield's (4) observations in western Mexico are of interest in this connection. He reported heavy infection on cotton 3.6 miles from the major site of overwintered grass and light infection at a distance of 8.4 miles. A reduction in the amount of grass near cotton fields would be beneficial, but elimination of the disease by eradication of the alternate hosts is not practical because of the wide grass-free zone required.

Rust damage was most severe on hard, woody cotton plants in the current epiphytotic. This has been observed by others (2, 4). In contrast to this, fields with plants of good vigor

FIGURE 2. A close-up of aecial pustules on an open boll showing a normal pustule above and one parasitized by Tuberculina below.

and leaf color escaped heavy losses even though general and severe rust occurred earlier in the season. It appears that steady, vigorous plant growth reduced rust damage by renewal of the foliage lost during periods of heavy infection.

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TRANSMISSION STUDIES OF HOJA BLANCA OF RICE IN COLOMBIA[1]

Guillermo E. Galvez E.[2], Peter R. Jennings[3], and H. David Thurston[4]

Summary

Hoja blanca is a virus disease transmitted by the leafhopper Sogata orizicola Muir. One single leafhopper can transmit the disease, but only about 9 percent of the natural population is capable of transmission. Adult males and females and nymphs transmit the disease. Hoja blanca was not transmitted through the soil or seed, or by mechanical means in these experiments.

Hoja blanca, a virus disease of rice, has recently been reported from Colombia, Cuba, Costa Rica, Panama, Venezuela, and the United States (2). Although the disease has received much recent attention owing to the serious reduction in rice yields in some of these countries, it has probably been present in Colombia since at least 1935 (3). The presence of a vector, Sogata orizicola Muir, has been confirmed (1, 4).

Gilberto Bravo, Agronomist of the Colombian Ministry of Agriculture's Entomology Program (unpublished data), was unable to transmit the disease with insects of the genera Draeculacephala, Neosteles, and Hortensia.

The present studies were initiated to look for a simple method of testing rice populations for disease reaction since field testing has often proven unreliable. Although their experimental materials and methods were not reported, Malaguti, et al. (4) mentioned that the disease was not transmitted through seed or by mechanical means. The following is a report of the results obtained from studies of transmission through seed, through the soil, by mechanical means, and by insects.

MATERIALS AND METHODS

These studies were begun in May 1958 at the Agricultural Experiment Station at Palmira, Colombia. All work reported was conducted in a screened greenhouse.

1. Soil Transmission: Soil collected from a field in which rice was severely attacked by hoja blanca was used to fill 30 pots. In each pot 10 seeds of the highly susceptible variety Bluebonnet 50 were planted. Germination was almost 100 percent. The test was made on four separate occasions.

2. Seed Transmission: Panicles showing symptoms of hoja blanca were selected from diseased plants from 50 susceptible varieties out of 2200 in an international rice nursery. The soil used for the experiment was disinfected with Bed-Drench. Fifteen seeds of each variety were planted in each of 10 pots. Average germination was 78 percent.

3. Mechanical Transmission: Leaves of plants of the susceptible variety Bluebonnet 50 were treated with juice extracted with a mortar and pestle from diseased leaves of the same variety. Care was taken to attempt inoculations immediately following extraction. The inoculations were made with: 1) a pestle, 2) a camel's hair brush, 3) gauze, and 4) the index finger. Each treatment was made with and without previous dusting of the leaves with number 320 carborundum powder. Ten pots with five plants each were used to test each method of inoculation, and with each treatment untreated check plants were included. The tests were conducted five times with plants 1, 2, and 3 months of age.

In other tests, roots and stems of plants 1 month of age were inoculated using a hypodermic needle filled with expressed sap of diseased leaves. Central leaf veins of 2-month-old plants were similarly inoculated. Ten pots with five plants each were used for each treatment. These tests included untreated checks and were repeated twice.

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[1] Paper No. 121 of the Agricultural Journal Series of the Rockefeller Foundation.

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4. Insect Transmission: Two sizes of transparent plastic cages were used, one for entire plants and the other for single leaves. In transmission tests both adults and nymphs of S. orizicola were allowed to feed for 3 to 4 days on infected caged plants; they were then transferred to healthy plants, where they stayed until death. In a test to determine the number of insects necessary to transmit the disease 1, 10, and 20 insects were used. Ten plants were infested with each number of insects.

RESULTS

1. Soil Transmission: In all tests no plant had symptoms of the disease after 60 days.

2. Seed Transmission: No plant grown from seed of infected panicles showed symptoms of the disease during 70 days' observation.

3. Mechanical Transmission: The tests using four methods of inoculation gave negative results at the end of 60 days or up to maturity.

The plants whose roots, stems, and central leaf veins were inoculated had negative readings after 40 days.

4. Insect Transmission: The only insect found to date to transmit the disease has been the leafhopper S. orizicola. In the test to determine the number of insects necessary to transmit the disease, groups of 10 and 20 insects gave positive transmission. A total of 500 adult insects of S. orizicola were tested individually, and of this number 43 were capable of transmitting the disease. Therefore, under the conditions at Palmira, Colombia, only about 9 percent of the natural population of the insect can transmit the disease. Both male and female adults and nymphs of S. orizicola were shown to transmit the disease. The ability to transmit appears to last throughout the life of the insect.

Symptoms were usually distinguished about 12 days after insects commenced feeding. Occasionally symptoms were observed after 5 days. In all cases symptoms appeared on leaves superior to, but not on, the inoculated leaf.

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COLOMBIAN MINISTRY OF AGRICULTURE, AND THE COLOMBIAN AGRICULTURAL PROGRAM OF THE ROCKEFELLER FOUNDATION

DEVELOPMENT OF THE ASPARAGUS RUST PATHOGEN IN RELATION TO TEMPERATURE AND MOISTURE

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Abstract

The development of the asparagus rust pathogen was studied under controlled conditions in the laboratory and greenhouse. Pycnial nectar and aecia appeared most rapidly on the stem between 25° and 30° C. The appearance of uredia was delayed for as long as 10 days by holding inoculated plants at 10° for 16 days but was delayed for a maximum of only 5 days when inoculated plants were first placed at 25° and then transferred to 10°, 15° or 20°. Primary uredia and rings of secondary uredia developed most rapidly above 25°. Maximum intensity of infection was obtained following 9 hours of continuous wetting at 15° although only a 3-hour period was necessary to obtain initial infection. Urediospores collected 5 to 7 days after rupture of the sorus germinated better at 10° or above than those collected earlier. Maximum germination of mature urediospores was attained in 3 to 4 hours at 10° to 15° C.

Rust, caused by Puccinia asparagi DC., is one of the most important diseases of asparagus, Asparagus officinalis. Some loss from this disease occurs each year in northern Illinois canning fields. This is especially true unless protective fungicides (7) are applied to young, unharvested fields which are the source of inoculum (urediospores) for the canning fields. Rust is of no importance in those areas of the State where asparagus is grown principally for the fresh market and where there are few if any young fields.

The effect of temperature and moisture on the development of asparagus rust has been studied by several investigators (3, 5, 6, 8, 9, 10, 11). With few exceptions (6, 8), conclusions have been based on field observations rather than on research under controlled conditions. Smith (9) observed dew to be essential for the development of rust and "without moisture of this sort no infection can take place, regardless of all other conditions." Free water is necessary for urediospore germination (6). Bremer (3) could find no significant correlation between meteorological conditions and the development of asparagus-rust epiphytotics in Germany, England, and the United States. However, Smith (9) claimed that once infection had taken place prolonged hot, dry periods delayed the development of aecia and subsequent spread of the disease. Fulton (5) found that maximum production of aecia in the field occurred when temperatures were between 19° and 22° C for several days. Sheldon (8) reported that under greenhouse conditions the incubation period following inoculation with urediospores was appreciably longer in December and January than in April and May at a time when temperatures were higher. The effect probably was one of temperature rather than light since light intensities from 14 to 6000 foot-candles have no influence on the length of the incubation period (2).

The present investigation was undertaken to determine the effect of temperature, moisture or a combination of these factors under controlled conditions on the development of aecia and uredia and on the germination of urediospores.

MATERIALS AND METHODS

One-month-old asparagus seedlings were transplanted into 2 1/2 inch clay pots in the greenhouse and allowed to produce three shoots, after which all were cut off at the soil line. The next shoot to appear was used for experimentation. Shoots to be used for inoculation were selected for uniformity in size and growth characteristics before they had "ferned out." The asparagus variety Carter's Mammoth Emperor was used in all experiments.

The urediospore culture used was obtained originally from an isolated sorus on a field-grown plant and increased in the greenhouse on Carter's Mammoth Emperor or on the variety

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[3] Professor of Plant Pathology Emeritus.

This study was conducted from July 1952 to March 1954 and was supported in part by Bankhead-Jones (Hatch) funds, Department of Horticulture Project 65-348.

Snowhead. Urediospores for subsequent study were collected from these plants by lightly tapping infected shoots over a large porcelain dish. Collections of spores not used immediately were stored at 2° to 5° C in sealed glass vials.

For uredial inoculations shoots of asparagus were sprayed lightly with distilled water, rubbed between moistened fingers to remove the waxy bloom and dusted with a 1:20 urediospore-talc mixture. The inoculated shoots were again sprayed lightly and incubated at 100 percent relative humidity in glass-walled moist chambers in the greenhouse or in constant-temperature chambers in the laboratory. The laboratory chambers were lighted with Mazda bulbs directly outside the glass front which provided a light intensity on the plants of 140 to 160 foot-candles. At the end of the incubation period, the plants were removed from the chambers, sprayed with tap water to remove the talc, and placed under the conditions provided for a given experiment.

For sporidial inoculations, presoaked teliospore-bearing asparagus stems were placed vertically alongside healthy shoots in moist chambers held at 18° to 20° C. Free moisture was maintained on the shoots by atomizing them lightly with distilled water.

Ratings for intensity of uredial development were made on comparable areas of the various shoots. The rating values were designated as: 0 = no infection; 1 = 1 to 5 sori; 2 = 6 to 15; 3 = 16 to 40; 4 = 41 to 75; and 5 = 76 or more. It was difficult to make ratings 12 days or more after inoculation because of coalescing sori.

Germination tests of urediospores were conducted at various temperatures on 0.5 percent distilled-water agar in Syracuse watch glasses. Two hundred or more spores were counted in each of two glasses for each treatment.

RESULTS

Temperature and the Development of Pycnia and Aecia: To determine the effect of temperature on the development of pycnia and aecia, shoots were inoculated with sporidia and left in the moist chamber for 40 hours. Six pots each containing a single shoot were then transferred to each of five constant-temperature chambers and held at 10°, 15°, 20°, 25° and 30° C. Observations on pycnial and aecial formation were made only on developing contiguous aecia, thus making it unnecessary to cross-transfer spermatia[4].

At 25° and 30° C, pycnial nectar and aecia appeared within 6 to 12 days after inoculation; at 20°, within 7 and 13 days; at 15°, within 8 and 20 days; and at 10°, within 13 and 29 days, respectively.

Low Initial Temperature and the Development of Uredia: In an experiment to determine the effect of low temperature on uredial development after inoculation, plants were inoculated and placed immediately in a constant-temperature chamber at 10° C. At 2-day intervals over a period of 16 days, four plants were removed and placed in a constant-temperature chamber at 25°.

The appearance of uredia was delayed appreciably by exposure to 10° C with the length of the delay directly proportional to the length of exposure. Uredia developed within 6 days on control plants placed at 25° immediately after inoculation. They were delayed 1 day on plants held at 10° for only 2 days, 5 days on those held for 8 days, and 10 days on those held for 16 days.

High Initial Temperature and the Development of Uredia: An experiment was conducted to determine the effect of high temperature after inoculation on subsequent development of uredia. Plants were placed immediately after inoculation in a constant-temperature chamber at 25° C. At daily intervals over a 5-day period, four plants were transferred to each of three constant-temperature chambers maintained at 10°, 15° and 20°.

Uredia were delayed in appearance by transferring plants to the three lower temperatures with the length of the delay inversely proportional to the length of exposure at 25° C (Table 1). Uredia appeared in 1 day after transfer of plants held for 5 days at 25° regardless of the temperature to which they were transferred. There was no retardation when plants were transferred from 25° to 10°, 15° or 20° after 5, 4 or 2 days, respectively.

Constant Temperature and the Development of Primary and Secondary Uredia: In asparagus rust, the fungus hyphae are limited at first to the tissue immediately surrounding the point of entrance of the urediospore germ tube. Later the pathogen spreads up and down the stem or branch producing successive rings of uredia. This phenomenon is similar to that described by Allen (1) for Puccinia graminis tritici Eriks. & E. Henn.

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[4] Aecia of P. asparagi develop from mating of haploid mycelia from contiguous stem lesions and also from cross-transference of spermatia (2).

To determine the effect of various constant temperatures on the development of primary and secondary uredia, shoots were inoculated with urediospores, and after 24 hours in the moist chamber five pots each containing a single shoot were transferred to each of four sections of a greenhouse maintained at 10° to 13°, 20° to 21°, 21° to 24°, and 26° to 28° C. Data were taken on the time of eruption of well-isolated primary uredia and rings of secondary and tertiary uredia.

Uredia developed most rapidly at 26° to 28° (Table 2). The time interval between the formation of secondary and tertiary uredia was somewhat shorter than that between the development of primary and secondary uredia.

Temperature and Moisture in Relation to Intensity of Uredia: Kahn, et al. (6) obtained a small increase in the number of uredia per plant by increasing from 15 to 40 hours the length of time plants were kept wet following inoculation. However, no attempt was made to control the temperature during incubation which was not specified. In the present studies, shoots were placed in moist chambers at 2°, 10°, 15°, 20°, 23° or 28° C for 8 hours prior to inoculation and were left there for periods of 1 1/2, 3, 4 1/2, 6, 9, 12 and 24 hours after inoculation. Distilled water, adjusted to the appropriate temperature, was atomized continuously on the plants before inoculation and during the incubation period. A total of 12 shoots were removed from each moist chamber at each of the time intervals cited and placed in a well-lighted section of the greenhouse at 26° to 28°. The intensity of infection was recorded 12 days following inoculation.

The minimum wetting period required to obtain initial infection was 3 hours, at temperatures of 15° C or above (Table 3). Maximum infection at this time interval occurred at 23°. Wetting for 9 hours at 15° resulted in the maximum value of 5.0 with progressively lower values at 20° to 28°. A period of 12 to 24 hours at 10° was necessary to reach near-maximum infection.

Temperature and the Germination of Urediospores from Sori at Different Stages of Maturity: A total of 10 plants in two tests were inoculated each day over a 7-day period. Records were kept on the date of sorus eruption and exposure of urediospores. Spores were collected from sori at all stages of development when the sori on the last group of plants inoculated had erupted. In this manner, a collection of spores from sori at seven stages of development was available for testing at one time. This procedure tended to minimize the factor of day-to-day fluctuation in percent germination. Duplicate germination tests for each stage of development were run at 5°, 10°, 15°, 20°, 25° and 30° C.

At 5° and 10° C there was no appreciable difference in germination between spores exposed 1 day and those exposed 7 days (Table 4). At all higher temperatures, germination increased with length of exposure; the highest being for those exposed 7 days. At 20° and above the over-all percentage declined. These results are in general agreement with those of Doran (4) who reported that newly-formed urediospores of Puccinia antirrhini Diet. & Holw. require near-optimum temperatures for good germination.

Temperature and the Rate of Germination of Urediospores from Mature Sori: Kahn, et al. (6) obtained maximum germination of urediospores at 20° C but did not determine the rate of germination at any of the temperatures employed. In the present investigation urediospores were collected 7 days after the sorus had ruptured and sown on temperature-adjusted water agar at 10- to 12-minute intervals. They were then placed at 2°, 5°, 10°, 15°, 20°, 25° or 28° C and left there for 1, 2, 3, 4, 6, 8, 12, and 24 hours. In this manner, the readings could be made at hourly intervals for the seven different temperatures.

At the optimum temperature range of 10° to 15° C for germination, the maximum percent germination was reached in 3 to 4 hours (Table 5). At 20° or above the peak was reached in 2 to 3 hours, but the percentage was much less. At temperatures below 10° maximum germination was attained in 6 hours or longer. There was no appreciable increase in the amount of germination at any temperature after 12 hours.

DISCUSSION

The asparagus-rust pathogen will develop over a fairly wide temperature range. Fulton (5) reported that teliospores germinate at temperatures between 10° and 26° C with an optimum of 20° and that the maximum production of aecia in the field occurs when mean temperatures are between 19° and 22° for several days. It was found in the present investigation that pycnia and aecia can develop over a range of 10° to 30° with an optimum of 25° to 30°. Urediospores germinate best at 10° to 15° although some germination occurs from 2° to 28°. Uredia develop over the same temperature range as do aecia and have approximately the same optimum. The development of uredia may be retarded as much as 10 days if the plants are exposed to 10° C

Table 1. Delay in the time of appearance of uredia on plants transferred at intervals from 25° C to three lower temperatures.

Days at 25° C	Delay[a] in appearance of uredia (days)		
	10° C	15°	20°
0 (Controls)	11[b]	4[b]	1[b]
1	9	3	1
2	7	2	0
3	5	2	0
4	2	0	0
5	0	0	0

[a] Calculated on basis of appearance of uredia in 6 days on plants held continuously at 25° C.
[b] Control plants placed at these temperatures immediately after inoculation.

Table 2. Length of time required after inoculation for development of primary, secondary and tertiary uredia at four temperature ranges.

Incubation temperature (° C)	Length of time after inoculation for appearance of uredia (days)		
	Primary	Secondary	Tertiary
10-13	17	24	31
20-21	8	15	18
21-24	6	12	15
26-28	6	10	13

Table 3. Effect of temperature and length of wetting period during incubation on intensity of uredia.

Hours of wetting	Mean intensity values at different incubation temperatures					
	2° C	10°	15°	20°	23°	28°
1 1/2	0	0	0	0	0	0
3	0	0	0.7	1.1	1.3	0.4
4 1/2	0	1.7	3.5	2.0	1.6	0.2
6	0	2.4	4.6	3.8	2.3	1.4
9	0	3.3	5.0[a]	4.7	2.7	2.0
12	0	4.8	5.0	4.5	2.6	1.6
24	1.2	5.0	5.0	4.4	2.3	2.1

[a] Maximum intensity value.

Table 4. Relation of temperature to percent germination of urediospores collected at four different intervals after sorus eruption.

Days after sorus eruption	Percent germination at different temperatures					
	5°C	10°	15°	20°	25°	30°
1	42	83	58	30	13	4
3	40	84	75	35	15	9
5	44	86	83	51	34	17
7	42	95	93	73	57	25

Table 5. Rate of germination of mature urediospores at seven temperatures.

Length of incubation (hours)	Percent germination						
	2° C	5°	10°	15°	20°	25°	28°
1	0	0	0	33	27	23	11
2	0	0	38	77	59	40	23
3	0	28	72	88	61	39	23
4	0	45	90	89	61	39	20
6	4	54	91	86	62	42	23
8	12	57	90	88	60	41	23
12	23	54	88	89	60	44	22
24	24	56	91	88	62	40	23

immediately after inoculation, but there is little retardation at 10° if the plants are exposed first to 25° for 4 to 5 days.

Temperature extremes that might be adverse to the pathogen do not prevail long enough in northern Illinois (Ogle County) to delay appreciably the ultimate development of epiphytotics. The mean daily air temperature[5] for May when teliospores and sporidia are germinating and the first aecia are developing is approximately 15° C with temperatures seldom over 30° or below 0°. The mean temperature for June when the primary uredia are forming is approximately 21°. Temperatures during the development of both aecia and primary uredia are usually below the optimum for growth of the pathogen although they are generally favorable for the production of uredia during the remainder of the growing season. The data from greenhouse experiments would suggest that the cumulative delay in the appearance of primary uredia in the field, brought about by below-optimum temperatures, might amount to as much as 2 weeks. Such a retardation is difficult to demonstrate in the field. It is likely then that an initial 2-week delay in the appearance of primary uredia in June would have little effect on the development of epiphytotics although it might benefit the grower by enabling him to postpone for 1 or 2 weeks the first application of fungicide to young, unharvested fields.

More favorable temperatures with a mean approximating 23° C prevail in July and August with the result that repeating cycles of urediospores are produced quickly and in abundance. Night temperatures of 12° to 18° during these months are favorable for urediospore germination. Germination of urediospores during the growing season is not likely to be severely limited by temperature extremes since only a short period of favorable temperature results in germination.

Moisture is not a limiting factor in the development of asparagus rust in Northern Illinois. Heavy dews occur frequently. This moisture, combined with a normal monthly rainfall of 3 to 5 inches for May through September, provides ideal conditions for germination of sporidia, aeciospores and urediospores. In addition, air drainage is generally poor in older fields, particularly after the middle of July when top growth becomes dense.

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[5] Mean monthly temperatures were calculated from U. S. Weather Bureau Climatological Data from 1949 to 1956.

PLUM RUSTY BLOTCH -- A TRANSMISSIBLE DISORDER FOUND IN SOUTHERN CALIFORNIA[1]

T. S. Pine and L. C. Cochran[2]

A disorder of Santa Rosa and Late Santa Rosa plum trees was first observed in 1953 in commercial orchards in San Bernardino and San Joaquin counties, California. Scions from affected trees were grafted to four healthy Santa Rosa trees in an experimental orchard in 1954. No transmission was noted in 1955, 1956, or 1957, although growth from the scions perpetuated the disorder. A few leaves below the grafts on the test trees showed mild symptoms during the growing season of 1958 but the number was not sufficient to permit a definite diagnosis. The first flush of growth in 1959 produced excellent symptoms on leaves of branches above and below the graft union. At the time of the original grafting, buds from affected Late Santa Rosa plum trees were placed in Lovell peach seedlings and healthy Late Santa Rosa buds were inserted about 2 inches above them. The peach seedlings were cut back to the healthy plum buds which were then forced into growth. The resulting trees showed no disease symptoms until the spring of 1959.

In 1958, a single branch of an otherwise healthy Santa Rosa tree in the experimental orchard which had been propagated as a cutting for other work showed symptoms of this disorder. Several other Santa Rosa and Late Santa Rosa trees growing either on their own roots or on myrobalan rootstocks showed varying amounts of symptoms during the summer of 1959. In some instances spread occurred to experimental trees which had previously been inoculated with the prune dwarf or line pattern viruses, but the presence of these viruses did not interfere with symptom expression of this new disorder.

The first symptom to appear on new spring growth of affected trees is a chlorosis which generally starts at the base of the leaf blade and extends toward the apex along one or both leaf margins (Fig. 1). This chlorosis tends to be continuous at first but may eventually take the form of chlorotic blotches. Within a few weeks the blotches become reddish brown and numerous small, red spots appear in the discolored areas. These spots may increase to about 1/8 inch in diameter or remain the size of a pinpoint, but they all eventually necrose and fall away, leaving the blotchy portions of the leaves irregularly shot-holed. The rusty blotch and necrosis may cover the whole leaf blade when leaves are severely attacked. Affected leaves are reduced to 1/3 to 1/2 the size of normal ones and tend to become misshapen as the growing season progresses. Young or old leaves of any branch may show the onset of symptoms concurrently, but by autumn all the leaves on an affected branch usually show infection.

In the orchard tree symptoms are generally observed first on a single branch. Because the leaves are discolored and reduced in size, these branches appear stringy and are readily noticed. Affected branches produce few blossoms and almost no fruit. Since initial symptoms are usually limited to a single branch, growers have been in the habit of removing these branches as they appear, but this practice has not contained the disease in the individual tree or in the orchard.

The disease has been seen in three separate orchards in San Bernardino County and in one orchard in San Joaquin County. Spread within each orchard is apparently slow. The orchard in which the original observations were made is now approximately 10 percent infected. No particular pattern of spread has been noted. Natural spread in the experimental plot occurred over a distance of 200 feet. Since spread occurred uphill, against the flow of irrigation water, it is not related to factors carried in water.

Noninfectious plum shot hole[3], which is widespread in southern California plum orchards, is readily separable from the disease reported here (Fig. 1). The noninfectious shot hole appears later in the season on mature leaves only, the initial spotting is brown or purple and occurs at random over the leaf blade, there is no reduction in leaf size and no leaf discoloration other than the incipient necrotic spots, there is no reduction in blossoming or fruit produc-

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[1]Cooperative investigations between Crops Research Division, Agricultural Research Service, United States Department of Agriculture, and University of California Citrus Experiment Station, Riverside, California.

[2]Plant Pathologist and formerly Plant Pathologist, respectively, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, and Research Associates, University of California, Citrus Experiment Station, Riverside, California.

[3]Smith, C. O., and L. C. Cochran. 1943. A noninfectious heritable leaf-spot and shot-hole disease of the Beaty Plum. Phytopathology 33: 1101-1103.

FIGURE 1. Three leaves on left from Santa Rosa plum trees affected with plum rusty blotch. Leaf on right from Santa Rosa tree with noninfectious plum shot hole.

tion, and the disorder has not been transmitted experimentally by tissue grafting. Noninfectious shothole and rusty blotch sometimes occur in the same tree but this coexistence does not interfere with the symptom picture of either condition.

The symptomatology and mode of transmission of this disease strongly suggests the presence of a virus. The name "plum rusty blotch" is proposed for the disease. Although the disease is not believed to be widespread, natural spread in orchards makes it of potential concern. Experiments are being conducted to determine the potential host range of the virus in other varieties of plum and in other Prunus species.

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CLITOCYBE MUSHROOM ROOT ROT IN GEORGIA[1]

J. H. Owen, J. H. Miller, and W. A. Campbell

Mushroom root rot caused by Clitocybe tabescens (Fr.) Bres. is a widespread and destructive disease in the southeastern United States, where the fungus has been found to attack over 200 hosts. The fungus was previously thought to be causing little or no damage in Georgia. In 1941 Rhoads[2] made a plant disease survey in southwestern Georgia and failed to find any evidence of the Clitocybe mushroom root rot disease. Savage, et al.[3] in 1952 reported the disease on peach trees in Georgia and stated that the fungus was probably well established and causing much loss to peach orchards throughout the State. Rhoads[2] only reported the disease in one or two instances on hardwood trees.

In October 1959 clusters of C. tabescens were produced abundantly at the bases of many living hardwood shade trees in the Athens area (Fig. 1A). The mushrooms were found attached to the crowns and roots of Magnolia grandiflora, Cornus florida, Ulmus americana, Quercus nigra, Q. phellos, Q. stellata, and Acer saccharinum. They were also frequently observed growing from the stumps and buried remains of hardwood trees in lawns and along the streets. Several woody areas in the vicinity of Athens were surveyed, and the clusters of mushrooms were found at the bases of numerous living and dead hardwood trees. Reports from other parts of Georgia indicated that C. tabescens sporulated abundantly all over the State in the fall of 1959. Dr. J. H. Miller stated that in his 40 years at the University of Georgia this was the first time that widespread sporulation of the fungus had occurred in Georgia.

A young peach orchard on the University of Georgia Horticultural Farm was found to be heavily infected by the fungus, and sporophores were produced at the bases of many of the infected trees (Fig. 1B). A 20-year-old peach orchard in nearby Watkinsville, Georgia -- reported by Rhoads[2] in 1954 as being infected by C. tabescens -- was visited during October 1959, and dried clusters of the fungus were observed at the bases of more than 50 percent of the trees in a 36-acre block. One-hundred percent of the trees in this planting were either dying back or were dead (Fig. 2). In all trees examined the white mycelial mat was revealed

FIGURE 1. A -- Cluster of mushrooms; B -- Sporophores of C. tabescens growing at base of infected peach tree.

[1] Approved as journal paper No. 118 of the College Experiment Station. The University of Georgia College of Agriculture Experiment Stations, Athens, Georgia.

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FIGURE 2. A 20-year-old peach orchard near Watkinsville, Georgia, showing trees killed by C. tabescens.

FIGURE 3. Infected trunks and crowns of peach trees girdled by C. tabescens, showing the white mycelial mat in the cambium area.

Table 1. Daily rainfall, Athens, Georgia, May through October 1959.

Day	Rainfall (in inches)					
	May	June	July	August	September	October
1	--	.99	.12	.10	.03	--
3	--	1.00	--	--	--	--
5	--	--	--	--	1.02	.37
7	--	.30	--	--	1.59	.06
9	--	--	--	--	1.77	.75
11	.30	--	1.77	--	.35	.12
13	1.24	--	2.65	--	.20	.86
15	.57	--	.16	--	.42	.61
17	--	--	1.57	--	.22	.35
19	.29	--	.81	--	--	--
21	.75	.32	1.66	.03	--	--
23	.02	.10	.52	.78	--	.87
25	.88	1.07	--	.02	--	.35
27	1.52	--	--	--	--	.05
29	.01	--	--	.08	--	.49
31	3.29	--	--	.20	.21	.27
Totals	8.87	3.47	9.26	1.21	5.81	5.34
Long-Time Mean	3.36	4.01	4.71	3.66	2.31	2.71

beneath the bark at the soil level (Fig. 3). An adjoining 7-year-old orchard of 36 acres was also found to have trees infected by C. tabescens, and about 15 percent of the trees in this orchard had died and been replaced. The white mycelial mat was observed under the bark of infected peach trees as high as 6 to 8 inches above the soil surface. Previous reports[2,3] indicated that this mycelial growth was usually 3 to 4 inches below the soil surface. The land where these three peach orchards are planted has been under cultivation of various field crops for over 40 years, and only the orchard located on the University of Georgia Horticultural Farm had been previously planted to peaches. There appeared to be no definite pattern of spread by the fungus in the young orchards, since diseased trees were found scattered throughout the orchards.

The rainfall for Georgia from May through October (Table 1) was much higher than normal, with the exception of June and August. The rainfall in September and October was more than twice the normal long-time mean, and usually these 2 months are relatively dry for Georgia. The excessive rainfall throughout the summer months, and particularly in September and October, no doubt accounted for the widespread sporulation of this fungus and possibly for the increased growth of the mycelium beneath the bark in the cambium area of the infected trees. When the sporophores were first observed in Georgia in 1952[3], the rainfall during September was reported as heavier than normal.

The abundant sporulation of this fungus during October is evidence of its widespread occurrence in Georgia (on many hardwood trees in addition to peach), and indications are that it is much more destructive than had previously been estimated. It probably attacks many other perennial plants, particularly ornamentals and shade trees, and should be considered a serious plant pathogen in this State.

While Armillaria mellea (Vahl) Quel. is the principal causal agent of mushroom root rot of trees in more northern States, it has not been reported on peach trees in Georgia.

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ADDITIONAL RECORDS OF VEGETABLE DISEASES IN ALASKA

Charles E. Logsdon and Gary Strobel[1]

The increasing interest in plant diseases in Alaska is reflected in the number of publications on the subject since 1950. Lefebvre (5) spent the growing season of 1948 in Alaska and recorded his observations in the Plant Disease Reporter in 1950. Sprague and Fischer (7) in 1952 issued a checklist of diseases of grasses and cereals which included Alaskan material. In 1953 Cash (1) compiled an excellent checklist of Alaskan fungi by bringing together many scattered records and by examining herbarium and fresh specimens.

Sprague (6) in 1955 combined records from his previous checklist (7), records from Cash's publication (1) and records of fungi he collected in Alaska in 1952 and published a separate checklist of diseases of cereals and grasses in Alaska. Dickson (2) further added to the list of diseases of grasses and legumes with his survey of 1956. Dickson and co-workers continued this survey in 1957, 1958 and 1959 and publication awaits identification of these specimens. Next was a report by Kimmey and Stevenson (3) on forest diseases in Alaska. Lebeau and Logsdon (4) in 1958 added three low-temperature pathogens to the growing list.

A number of economically important diseases of Alaskan vegetables have either not previously been observed or if observed, they have not been previously recorded. The following report lists 22 vegetable diseases in Alaska for which the authors can find no published record. The majority of these diseases were noted during the 1959 growing season which was extremely favorable for disease development because of almost continual rain from June 20 to September 1.

Bean: Pellicularia filamentosa was found at Palmer, on bean stems just above the ground line. It apparently was doing no damage, and there was little damage from Rhizoctonia on the underground portions.

Sclerotinia sclerotiorum was found also at Palmer, attacking the stems and pods of individual plants. Sclerotia were abundant on the surface of the host tissue but were produced only sparingly on PDA.

Cabbage: Fusarium was the most important cause of damping-off this season at Palmer. This organism was not identified to species.

Hormodendrum was isolated from the outer leaves of cabbage growing at Fairbanks, but it was thought to be either secondary or saprophytic.

Rhizoctonia solani was serious on a field of direct-seeded cabbage near Fairbanks, where it stunted the plants and prevented heading.

Soft rot (probably Erwinia carotovora) of the roots and lower stem of cabbage was almost universally associated with plants attacked by root maggots. Isolations from surface-disinfected maggots yielded pure cultures of this bacterium.

Carrot: Botrytis cinerea appears to be the most important storage disease of carrots in the Matanuska Valley. It attacks the side rootlets as well as the tip of the main root and develops slowly throughout the period of storage at near freezing temperatures.

Stemphylium radicinum is a very common storage disease in the Matanuska Valley, causing black rot of the crown. Inoculation of the soil with this organism immediately after planting caused severe cracking of the crown in the harvested carrots, as well as extensive black rot in storage.

Celery: Blackheart, a physiological problem, was present but not serious this year in the vicinity of Palmer.

Late blight (Septoria apii-graveolentis) was present on many plants in the Wasilla area. It was serious enough after 1 month in storage to cause considerable grade-out loss. Most celery grown in Alaska is shipped in as transplants and this may account for the presence of this disease in Alaska.

Lettuce: Isolations from seedlings at Palmer that were damping-off yielded Fusarium. This has not yet been identified to species.

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Downy mildew (Bremia lactucae) was severe enough in one 3-acre field near Sutton to cause the grower to abandon the crop.

Onion: Fusarium sp. was isolated from damped-off seedlings at Palmer.

Botrytis cinerea grew profusely on the leaves of green onions in storage at Palmer.

Peas: Alternaria found on leaves and stems of peas at Matanuska was probably secondary or saprophytic, since it attacked only the older portions that were chlorotic from lack of sunshine. This was not identified to species.

Fusarium was isolated from the root and crown area of peas in a Fairbanks garden. This tentatively has been identified as Fusarium solani f. pisi.

Pepper: Botrytis cinerea severely attacked peppers in the experiment station greenhouse at Palmer in 1959. Primary infection occurred on the blossoms or on the flower parts adhering to the young fruit. From this start, it spread into the stems. The fungus also attacked older fruits, without growing through the flowers, producing more or less circular sunken lesions. There was no evidence that the organism was secondary or that infection had come from infected floral parts falling on the plant.

Potatoes: Button rot (Phoma tuberosa) was identified from the Fairbanks area in 1956, where it caused considerable grade out at that time. It has been of no particular concern since then.

Rhizoctonia violacea was isolated from typical Rhizoctonia lesions on potato stems in the Fairbanks area. This tentative species identification is based on the purple pigment produced in culture, which is readily removed with a solution of HCl.

Skin spot (Oospora pustulans) was found in the Wasilla area, on stored potatoes in the spring. The disease was very severe in at least one lot of potatoes this year in the Fairbanks area, where it has been an increasing problem since 1954.

Radish: Soft rot (probably Erwinia carotovora) was quite common in radishes infested with root maggots.

Rhizoctonia solani was isolated from radishes where it caused water-soaked sunken lesions about three-fourths or more the size of the diameter of the radish. These lesions were found after about 2 weeks' storage although they were not apparent at harvest.

It seems obvious from the foregoing list that plant diseases are increasing in numbers and importance throughout Alaska as agriculture in the new State expands. Although plant diseases have been of minor importance in the past, they now promise to become a major limiting factor in crop production in Alaska.

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THE SEPARATION OF NEMATODES FROM SOIL BY A MODIFIED BAERMANN FUNNEL TECHNIQUE[1]

J. T. Walker and J. D. Wilson[2]

Summary

A modification of the Baermann funnel technique was devised to permit the collection of nematodes from soil samples without repeated sievings or elaborate soil processing methods. The device consists of a special container to hold the soil samples in the flaring portion of a funnel. Comparative data on the yields of nematodes from three different soil types using this modified Baermann funnel technique and the Seinhorst elutriator are presented. With silt loam soil, the modified Baermann funnel technique yielded 16 percent as many nematodes as the elutriators; with muck soil, 60 percent; and with a clay loam about the same number. The percentage of stylet-bearing nematodes was greater from muck and silt loam samples obtained by the modified Baermann technique. Of the plant parasitic forms separated from the silt loam soil, the percentages of Paratylenchus spp. and cyst-forming types were higher in those suspensions obtained with the funnel technique, and lower for Pratylenchus spp., Helicotylenchus spp., and Criconemoides spp.

If settling time was disregarded, this alteration of the Baermann funnel technique permitted populations to be determined in about 1/12 the time required by the elutriator method.

INTRODUCTION

The most practical and efficient means of separating nematodes from soil samples or plant material has been the concern of nematologists for many years. A multiplicity of methods have been described (1, 2, 4, 7), each possessing merits that warrant its use for specific purposes. The Baermann funnel technique, with its numerous modifications, has been one of the most widely used in spite of certain inherent weaknesses. The technique is easy to use and is capable of furnishing specimens comparatively free of foreign material. However, the quantitative yields may be variable, depending partly on the size and character of the samples placed in the funnel (3).

Since the Baermann funnel technique offered the quickest possible means of examining a large number of soil samples in an extensive crop rotation study, it was chosen as the most practical method of checking monthly variations in nematode populations. The purpose of the present paper is to introduce a modification of this technique which further speeds up the handling of samples. Data obtained with this modified technique as compared with the Seinhorst elutriator method (5) are presented.

METHODS

The present modification of the Baermann funnel consists in providing a screened container for the soil sample. This device, similar to that described by Staniland (6) and others (1), consists of a 2-inch section of aluminum irrigation pipe (3-inches in diameter), into one end of which a disc of 2-mm mesh aluminum screen is tightly held by a band of aluminum. The assembled container and components are shown in Figure 1. A double thickness of wet strength tissue (Hudson Hankies were used in this study) 6 inches square was placed on the screen and a level tablespoon (approximately 15 cc) sample of soil placed on the tissue. The container was placed in the flaring portion of the funnel and water added in an amount sufficient to wet but not flood the soil. After 24 hours, 10 ml of suspension was drawn from the bottom of the funnel. From this suspension four aliquots were placed in a Fenwick multichamber counting slide. Nematode counts were made for each aliquot with a stereoscopic microscope. These data were converted to give the corresponding number of nematodes per pint of soil (approximately 500 cc). In making the comparative counts mentioned below, nematodes separated from soil by means of Seinhorst elutriators were suspended in known volumes of water and counted in the same manner as those from the funnels.

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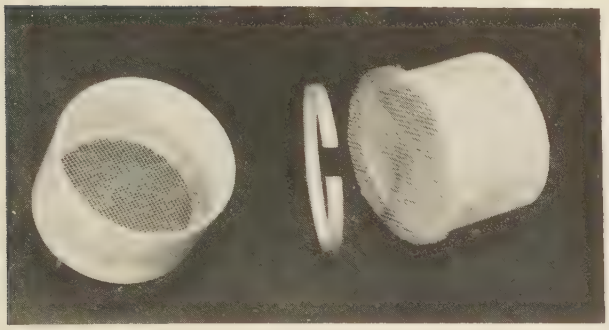

FIGURE 1. Assembled container for soil samples on left, and the three components before assembly on the right.

In an effort to compare the recovery of nematodes from soil by the two methods, samples were collected from three soil types (clay, muck, and silt loam). Three samples of 32 borings in each were removed from a plot 10 feet square of each soil type. Borings were made with a 1-inch diameter sampling tube to a depth of 6 inches. Each sample was subsequently mixed in a portable cement mixer, after which one-tablespoon quantities were placed in each of eight aluminum containers and 1- (approximately 250 cc) and 2-cup quantities were processed in two Seinhorst elutriators. The nematode suspensions recovered from the Seinhorst apparatus were passed five times through the recommended soil sieves (5).

The silt loam plot was in a lawn area covered primarily with blue grass (Poa pratensis), and white clover (Trifolium repens). The muck area had been planted earlier in the season to celery and onions, and a crop of corn had been growing on the clay plot.

RESULTS

More nematodes in a given quantity of soil were recovered from the elutriated samples of silt loam and of muck than from those recovered by the modified Baermann funnel technique (Table 1). The greatest difference in number of nematodes recovered by the two methods was with the silt loam, where the recovery was approximately six times as great by the Seinhorst elutriator. Less difference occurred with muck soil; the recovery was approximately 40 percent more in the elutriated samples. With the clay soil, the number of nematodes recovered was about the same by the two methods. The variation in yield among all samples was less by the modified Baermann funnel technique than by the Seinhorst elutriator (Table 1), which may permit the use of a smaller number of replicate samples.

The total time involved in processing the soil samples by the two methods was considerably different, as is indicated in Table 1. In most instances the time necessary to carry out the procedure using the modified Baermann funnel technique was only about one-twelfth that with the Seinhorst elutriator.

Table 1. A comparison of numbers of nematodes recovered from soil by means of a modified Baermann funnel and elutriator methods, and the time required for each method.

Soil type	Nematodes recovered[a]				Time required[b] (minutes)
	Sample 1	Sample 2	Sample 3	Average	
Mod. Baermann funnel:					
Clay	2280	2680	2180	2380	29
Muck	460	330	240	343	39
Silt loam	1140	1210	1240	1197	29
Seinhorst elutriator:					
Clay	2024	3041	1875	2313	373
Muck	874	294	333	500	557
Silt loam	7066	4708	10645	7473	316

[a]Average number of nematodes per pint based on eight determinations for each sample in the modified Baermann funnel method, and on two determinations for each sample in the Seinhorst elutriator method.

[b]Exclusive of 24 hour settling time for the modified Baermann funnel method.

The number of stylet-bearing nematodes from each sample was recorded separately and the percent for each soil type is presented in Table 2. The recovery of nematodes by the modified Baermann funnel technique was obviously dependent upon the motility of the nematodes, whereas this was not so when the Seinhorst elutriator was used. Thus, the percentages of stylet and saprophagous forms would be expected to be different in the populations recovered by the two methods. The data presented indicate that the proportion of stylet-bearing nematodes in the muck and silt loam samples processed by the modified Baermann funnel technique was greater than those recovered from these samples by the Seinhorst elutriator.

Table 2. Percent of stylet-bearing nematodes in samples from three soil types as determined by the modified Baermann funnel and Seinhorst elutriator techniques.

Soil type	Modified Baermann funnel	Seinhorst elutriator
Clay	34.5	38.5
Muck	44.6	38.0
Silt loam	52.9	48.4

The diversity of plant parasitic genera was found to be greatest from the silt loam soil and for this reason it was chosen for comparing the numbers of stylet-bearing nematodes in relation to the total recovered by each method. The data obtained are given in Table 3.

Table 3. Proportion of different kinds of stylet-bearing nematodes to the total stylet-bearing nematodes recovered from silt loam soil by two methods.

Recovery method	Percentage of each kind of nematode				
	Cyst forming	Lesion	Pin	Ring	Spiral
Modified Baermann Funnel	35.2	4.2	2.1	0.0	5.7
Seinhorst Elutriator	9.4	9.1	1.6	3.7	15.8

Cyst (Heterodera spp.), lesion (Pratylenchus spp.), pin (Paratylenchus spp.), and spiral (Helicotylenchus spp.) nematodes were separated by both methods. No ring nematodes (Criconemoides spp.) were obtained by the modified Baermann funnel technique, although they were present in the suspensions obtained with the Seinhorst elutriators. The larger number of larvae of the cyst-forming nematodes obtained by the modified Baermann funnel technique may have been due to the rapid hatching of eggs during the settling time. It should be noted that in the other two soil types the genera which were present in the suspensions derived from the funnels were also present in the suspensions derived from the elutriators.

DISCUSSION

The use of the soil container as an adjunct to the Baermann funnel technique facilitated the recovery of nematodes when a large number of soil samples were examined. The use of this container for supporting the soil sample in the funnel lessened the chance of soil spilling into the funnel and thus increased the cleanliness of the nematode suspension. Freedom from foreign material in the nematode suspensions permitted faster counting. The soil container can be constructed with inexpensive materials.

Although fewer nematodes were recovered from certain soils by this modified Baermann funnel technique than by the Seinhorst elutriator method, the nematode suspensions obtained by the use of the former technique were judged to be fairly reliable in comparing the nematode populations in crop rotation studies. The small variation in number of nematodes from sample to sample in the altered Baermann funnel technique suggests that a statistical comparison with other methods, using a larger number of replicates, may be worthwhile.

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ADDITIONAL HOSTS FOR TOMATO CANKER ORGANISM, CORYNEBACTERIUM MICHIGANENSE

Peter A. Ark and James P. Thompson[1]

Abstract

Solanum muricatum and Solanum mammosum, both perennial weeds, are found to be susceptible to Corynebacterium michiganense. They appear to be new hosts.

It has been shown that Solanum douglasii, a native perennial, can be infected by the tomato canker organism, Corynebacterium michiganense. Diseased plants of this species were found in the vicinity of a tomato field[2, 3].

When the senior author was in Australia in 1951, he learned from Mr. W. C. Sutton, of the Division of Plant Pathology of the New South Wales Department of Agriculture, in Sydney that a wild Solanum nigrum can occasionally be found harboring the tomato canker organism. The importance of weeds in harboring potential plant pathogens cannot be underestimated. Therefore, different species of weeds grown from clean seed in the greenhouse have been inoculated with Corynebacterium michiganense. Two species, S. muricatum and S. mammosum, have been found susceptible to C. michiganense. S. mammosum is grown as an ornamental plant in Honduras. S. muricatum is known as pepino in Guatemala and is occasionally grown for its fruit.

FIGURE 1. Symptoms on Solanum muricatum inoculated with Corynebacterium michiganense and held in warm greenhouse (70° to 80° F) for 30 days. Note wilting of the upper leaves. Inoculation made by decapitating the plant with a sharp scalpel contaminated with the pathogen.

[1] Professor of Plant Pathology and Laboratory Technician II, respectively, Department of Plant Pathology, University of California, Berkeley, California.

[2] Baines, R. C. 1947. Perennial nightshade, a host for Corynebacterium michiganense. (Abst.) Phytopathology 37: 359.

[3] Haven, Lawrence. 1945. Nightshade source of tomato infection. Seed World 57: 18.

FIGURE 2. Symptoms on Solanum mammosum following a wound inoculation at the top with Corynebacterium michiganense. The plant was held for 30 days at 70° to 80° F. Note wilting of the upper and middle leaves.

Solanum muricatum appears to be extremely susceptible to Corynebacterium michiganense and typical tomato canker wilt symptoms appear within 11 to 14 days after inoculation (Fig. 1). S. mammosum survives infection longer than S. muricatum (Fig. 2).

DEPARTMENT OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA, BERKELEY, CALIFORNIA

A METHOD TO INDUCE WOODY GALLS ON DECIDUOUS FRUIT TREES WITH AGROBACTERIUM TUMEFACIENS AND ON OLIVE TREES WITH PSEUDOMONAS SAVASTANOI

Peter A. Ark and James P. Thompson[1]

Abstract

Woody galls can be produced on potted apricot and peach trees by inoculating with a young agar culture of Agrobacterium tumefaciens using Swiss pattern round files (5 1/2 or 7 3/4 inches long) and filing the living tissue to the cambium layer. When the same technique is applied to olive trees using Pseudomonas savastanoi, large knots are produced.

Much of the inoculation work on crown gall reported in the literature is based on soft galls produced on herbaceous plants such as tomato (Lycopersicum esculentum), sunflower (Helianthus annuus), and chrysanthemum (Chrysanthemum hortorum). Some phases of crown gall work, the development of eradicant chemical treatments, for example, require the use of hard galls such as occur in apricot or peach orchards. A classical needle inoculation is successful only when it is performed on young tender shoots. Under these conditions comparatively small soft galls are produced which, although becoming harder in time, usually grow very slowly and for certain experimental procedures are not comparable to those occurring in orchards. A

FIGURE 1. Crown gall tumors induced by the file method. A -- Plant bearing many galls variously located. Leaves were removed for better photographing. B -- Galls on middle of the plant in A; C -- one of the galls enlarged.

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FIGURE 2. Crown gall on lower part of a tomato plant (Bonnie Best variety) induced by inoculation with Agrobacterium tumefaciens, using the file method. Two-month-old growth.

simple but efficient way of avoiding this difficulty is to use a small size (5 1/2 or 7 3/4 inches long) Swiss pattern round file to make the wound and a heavy aqueous suspension of crown gall organism, Agrobacterium tumefaciens.

Inoculation is accomplished either by filing the plant tissue with a file that has been dipped in a suspension of the organism, or by filing first with a clean file and then smearing the wound with a bacterial suspension. Filing is continued until the cambium of the shoot is exposed; the larger the cambium strip thus exposed, the larger the resulting gall. However, an excessively large wound may not be desirable since it can cause a wound reaction of the plant (gumming) and there is greater danger of tissue desiccation unless the wound is held for a considerable time under 60 to 70 percent relative humidity. Usually a little practice with the method is sufficient for uniformly successful production of woody crown galls or olive knots (Fig. 1).

When this method was used on large tomato plants (Bonnie Best variety) huge galls were produced in a relatively short time (Fig. 2). For crown gall infection the cambium layer is the most susceptible part of the plant.

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SUSCEPTIBILITY OF ARTEMISIA VULGARIS AND HELIANTHUS TUBEROSUS TO CROWN GALL, AGROBACTERIUM TUMEFACIENS

Peter A. Ark and James P. Thompson[1]

Abstract

Artemisia vulgaris and *Helianthus tuberosus* produce rapidly growing galls when inoculated with *Agrobacterium tumefaciens*. The plant are deemed to be new hosts for *A. tumefaciens*.

Knowledge of new suscepts of *Agrobacterium tumefaciens* is valuable from the standpoint of a possible use for such plants in crown gall research. For example, the susceptibility of carrot root slices[2] has proved useful in detecting *A. tumefaciens* in infested soils.

Sagebrush (*Artemisia vulgaris*) grows well under greenhouse conditions, and can be propagated with ease from seed and from either green or hard cuttings. Stem and root inoculations (by needle or file methods) with *Agrobacterium tumefaciens* result in visible tumorous growth within 3 or 4 days at temperatures of 70° to 80° F (Figs. 1, 2). When green cuttings of *A. vulgaris* are dipped in a suspension of *A. tumefaciens* and set in moist coarse sand, tumor tissue develops on the cut surface within 2 or 3 weeks.

Equally successful results are obtained when tubers or semi-hard cuttings of Jerusalem artichoke (*Helianthus tuberosus*) are inoculated by the methods used for *Artemisia vulgaris* (Figs. 3, 4). *Helianthus tuberosus* is propagated by tubers after a rest period and by cuttings continually throughout the year.

While *Helianthus annus* is a well known host of *Agrobacterium tumefaciens*, no reference has been found to crown gall on *H. tuberosus* and *Artemisia vulgaris*.

FIGURE 1. Crown gall on *Artemisia vulgaris*. Inoculation was made by puncturing the stem with a sharp dissecting needle charged with a 24-hour-old bacterial growth of *Agrobacterium tumefaciens* on potato-dextrose-peptone agar. One month's growth.

[1] Professor of Plant Pathology and Laboratory Technician II, respectively, Department of Plant Pathology, University of California, Berkeley, California.

[2] Ark, Peter A., and Milton N. Schroth. 1958. Use of slices of carrot and other fleshy roots to detect crown gall bacteria in soil. Plant Disease Reptr. 42: 1279-1281.

FIGURE 2. Crown gall on root of Artemisia vulgaris induced by a needle inoculation with a pure culture of Agrobacterium tumefaciens. The plant was placed in sterilized moist sand for the duration of the experiment. Thirty days after inoculation.

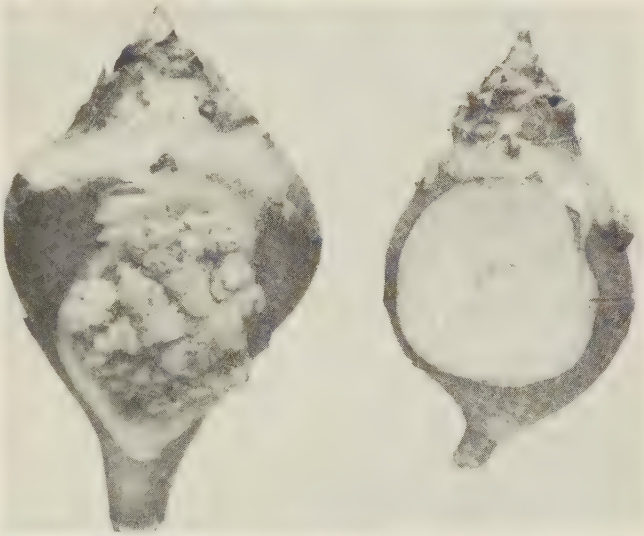

FIGURE 3. Left -- Three-week-old crown gall infection on cut surface of a tuber of Helianthus tuberosus. The tuber was sliced about 1/4 inch from the top and a suspension of a young agar slant growth of Agrobacterium tumefaciens was smeared on the freshly cut surface by means of a sterile cotton swab.

Right -- Uninoculated tuber.

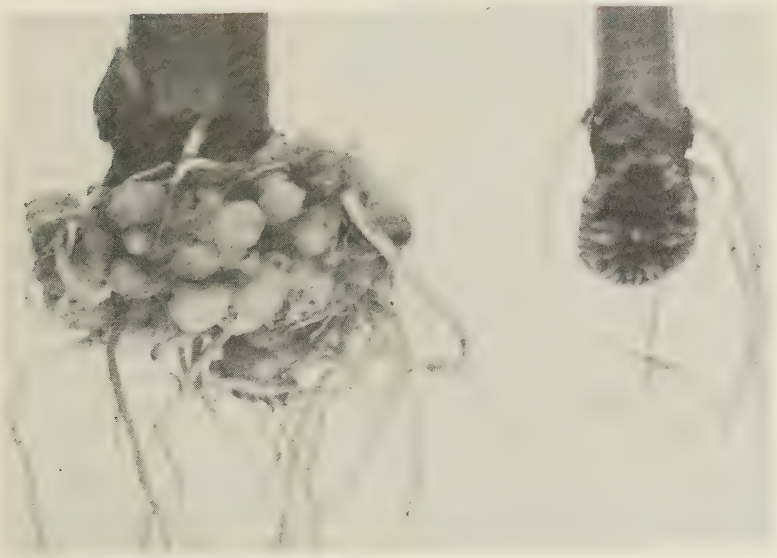

FIGURE 4. Left - One month's growth of crown gall on a cut surface of Helianthus tuberosus. Infection obtained by placing the cutting in sterilized sand contaminated with Agrobacterium tumefaciens.

Right -- Cutting from the series of similar cuttings set in non-contaminated sand and held in the same greenhouse for the same period of time as the inoculated set.

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EFFECT OF INSECTICIDE-FUNGICIDE COMBINATIONS ON EMERGENCE OF PEAS AND GROWTH OF DAMPING-OFF FUNGI[1]

Lloyd T. Richardson[2]

Summary

Pea seeds treated with commercial preparations of aldrin, dieldrin, or lindane, singly or in combinations with thiram, captan, or chloranil, were planted in sterile sand artificially infested with a series of concentrations of inoculum of Pythium ultimum Trow or Rhizoctonia solani Kuehn. In the absence of these pathogens none of the treatments had any effect on the emergence of seedlings. In infested soil, however, seeds treated with insecticides alone were more vulnerable to pre-emergence damping-off than untreated seeds, whereas seeds treated with insecticide-fungicide combinations were better protected than those treated with fungicides alone. The insecticides were also added, with and without the fungicides, to culture media inoculated with the same fungi. By themselves the insecticides (particularly lindane) retarded mycelial growth, and in combination they added to the inhibitory effect of the fungicides.

Improved emergence of seedlings has been reported by various investigators (2, 3, 4, 5, 7, 9, 12) to result from various insecticide-fungicide combination seed treatments as compared with single treatments with either insecticides or fungicides. Since both insects and fungi were generally present in the soil, however, these tests gave no indication of what effect the insecticides themselves might have on pathogenic soil-borne fungi or on the incidence of pre-emergence damping-off. Consequently a study was made on the effect of various chlorinated hydrocarbon insecticides, alone and in combination with fungicides, on the emergence of pea seedlings planted in sand infested with pure cultures of damping-off fungi. The effect of the insecticides, with and without fungicides, on the growth rate of these fungi was also investigated in an attempt to account for observed effects on disease development.

METHODS

Commercial preparations of various insecticides and fungicides commonly used for seed treatments were applied to Perfection pea seeds as a slurry suspended in 0.5 percent methyl cellulose[3] solution. The rates of application were equivalent to 1/4 ounce per bushel for 25 percent wettable powders of the insecticides aldrin, dieldrin, and lindane, and 1 1/3 ounce per bushel for the fungicides thiram[4], captan[5], and chloranil[6]. Treated seeds were planted at a uniform depth in waxed paper cartons filled with sterile river sand infested to various degrees by mixing with a series of proportions (by weight) of inoculum of Pythium ultimum or Rhizoctonia solani grown on sand-corn meal medium. Four replicates of 25 seeds each were used for each treatment. The seeds were incubated for 10 days at 70° F and 60 percent relative humidity under artificial light in a plant growth chamber. Emergence counts were then taken and the percentage pre-emergence kill was calculated from the difference between treatments and controls.

The plate culture method was used to determine the effect of the insecticides and fungicides on the growth rate of the fungi. All insecticides were tested at 25 ppm, a concentration inducing maximum inhibition of mycelial growth as shown by Richardson and Miller (10). The required amount of each compound, in the form of a commercial wettable powder, was first suspended in sterile 0.5 percent methyl cellulose solution, then pipetted into melted Czapek's agar at 50° C. A 4-mm disk cut from the margin of a 2-day-old plate culture of P. ultimum or R. solani was used as inoculum. Degrees of inhibition could not be determined from colony diameters measured at a stated time because some colonies showed no measurable growth before others had covered the plate. As a basis for comparison, therefore, the time (in days) re-

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[3]"Methocel", Dow Chemical Company.
[4]Arasan SFX
[5]Orthocide 75W
[6]Spergon SL

quired for a colony to reach a diameter of 80 mm was read from a growth curve derived from daily measurements. The growth lag was then calculated as the difference in this time period between treatments and controls.

RESULTS

The emergence of seedlings from insecticide-treated pea seeds planted in sterile sand was in no case less than the emergence from untreated or fungicide-treated seeds. The insecticide treatments, therefore, did not affect the viability of the seed or the pre-emergence development of the seedlings in the absence of a pathogenic fungus.

In sand infested with either Pythium ultimum or Rhizoctonia solani, insecticide treatments had opposite effects on emergence, depending on the presence or absence of a fungicide. When an insecticide alone was applied the percentage emergence from treated seeds was less than that from untreated seeds. When both an insecticide and a fungicide were applied the percentage emergence was greater than that from seeds treated with fungicide alone. The protective action of a fungicide was thus enhanced by the addition of an insecticide. The emergence of seedlings from seeds treated with lindane with and without thiram and planted in soil infested with P. ultimum is illustrated in Figure 1.

FIGURE 1. Emergence of seedlings from untreated pea seeds and seed treated with thiram, lindane, and thiram-lindane combination before planting in sand infested with Pythium ultimum. Inoculum levels, left to right: 0, 20, 10, 5, 2.5 and 1.25 percent by weight.

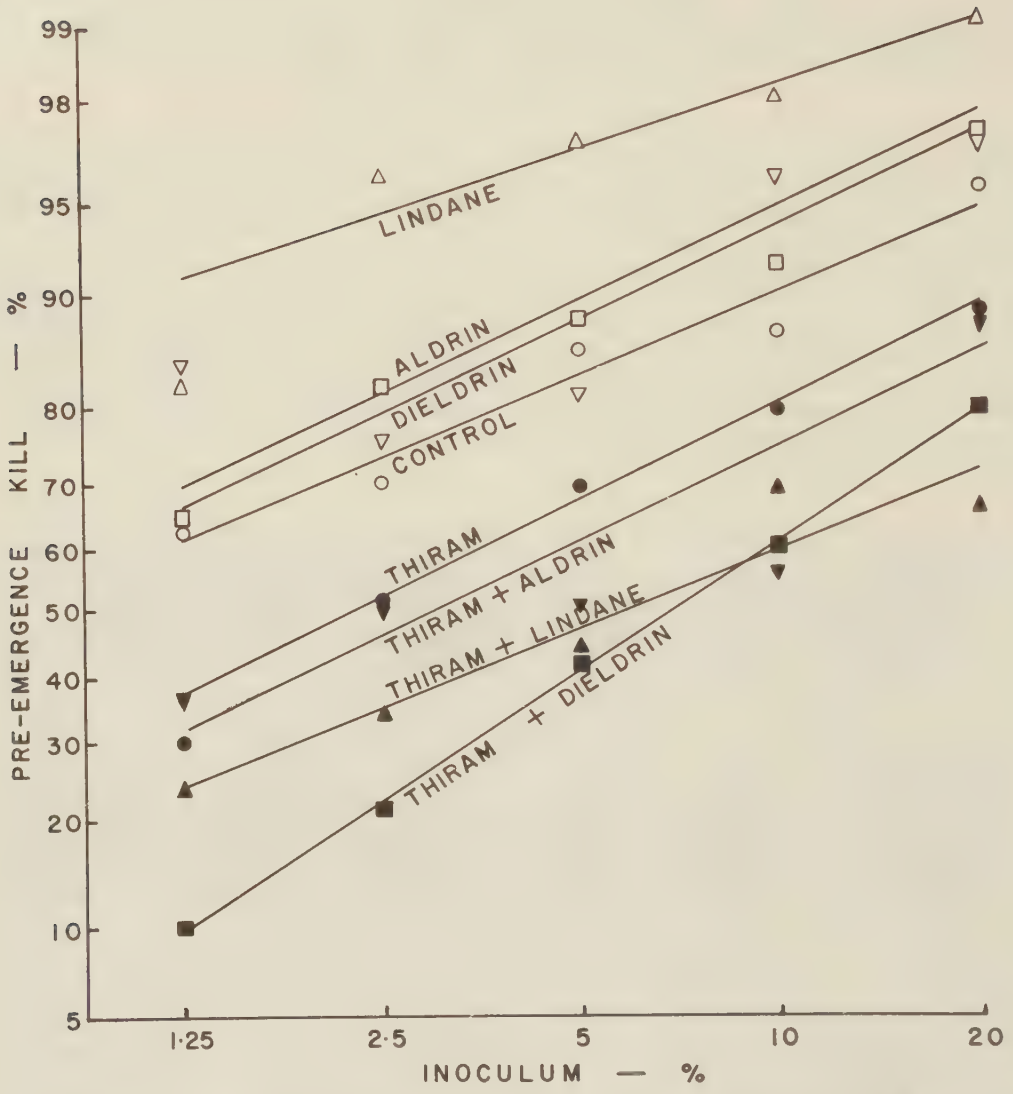

FIGURE 2. Effect of various seed treatments on incidence of pre-emergence damping-off of pea seedlings grown in sand infested with a series of concentrations of inoculum of Pythium ultimum. Symbols: circles -- no insecticide; squares -- dieldrin; erect triangles -- lindane; inverted triangles -- aldrin; solid symbols indicate thiram treatments.

A linear relationship between percentage pre-emergence kill and inoculum concentration was found when these data were plotted on probability paper. The relative positions of the curves obtained in this way following treatment with aldrin, dieldrin, and lindane, singly and in combination with thiram, are shown in Figure 2. All curves for insecticide treatments alone lie above the control curve, whereas all curves for combination treatments lie below the thiram curve. These results indicate that the incidence of pre-emergence damping-off is increased when an insecticide alone is applied to seeds, but the protective action of the fungicide is enhanced by the addition of an insecticide. Similar results were obtained when pea seeds treated with the same insecticides in combination with captan or chloranil were planted in sand infested with P. ultimum or R. solani.

Since the insecticides did not directly affect the viability of pea seeds, their effect on the development of the pathogens was investigated as a possible explanation of the differences in emergence due to seed treatments. The rate of mycelial growth of both fungi was found to be retarded by the presence of an insecticide in the medium, the greatest lag being induced by

lindane. Combinations of insecticide with a fungicide caused a greater lag than did the fungicide alone. Certain combinations were lethal at the higher fungicide dosages, as demonstrated by transferring inoculum disks that failed to develop to untreated medium. The lag periods found when P. ultimum and R. solani were grown on media containing aldrin, dieldrin, and lindane combined with a series of thiram dosages are given in Table 1. Similar results were obtained with these insecticides combined with captan and chloranil.

Table 1. Effect of various insecticide-fungicide combinations in culture medium on growth rate of two damping-off fungi.

Fungus	Insecticide (25 ppm)	Thiram (ppm) 0	1.5	3	6	12	24
		Lag period[a] (days)					
Pythium ultimum	None	0	0.5	1.3	2.3	4.5	6.8
	Aldrin	0.1	0.9	1.9	3.3	5.8	7.2
	Dieldrin	0.2	1.2	2.0	2.7	5.0	7.0
	Lindane	6.2	7.0	8.1	8.5	x[b]	x
Rhizoctonia solani	None	0	1.0	2.2	2.8	3.5	6.5
	Aldrin	0.6	2.9	3.5	3.3	5.4	x
	Dieldrin	0.6	2.9	3.0	3.3	6.4	8.5
	Lindane	2.5	3.1	3.7	4.6	6.1	x

[a]Additional time, in days, required by colonies on treated media to reach diameter of 80 mm as compared with colonies on untreated medium.
[b]Organism killed by treatment.

DISCUSSION

From the results of these trials it is evident that combination seed treatments can provide better protection than either insecticide or fungicide treatments alone, even in the absence of insects. The presence of a fungicide prevents possible increase in seed decay and pre-emergence damping-off by fungi induced by insecticide treatment. The presence of an insecticide can increase the effectiveness of a fungicide in controlling the disease.

Increased pre-emergence killing following insecticide treatments without a fungicide has been reported by various workers (2, 3, 4, 5, 7, 8) but the cause of the reaction has not been definitely established. Ditman et al. (2) suggested that injury to the pericarp by lindane facilitates invasion of the seed by fungi. It is also possible that insecticides have a physiological effect on the host, predisposing it to infection. Another possibility is that the insecticide neutralizes or prevents the formation of substances by the host which would normally inhibit the pathogen.

Chlorinated hydrocarbon insecticides do not invariably increase the incidence of seedling disease in the absence of a fungicide, however, according to reports in the literature. Arnold and Apple (1) found a low order of protection with dieldrin applied without a fungicide to corn seed planted in Pythium infested soil. Moreover, Simkover and Shenefelt (11) were able to control damping-off of pine seedlings by applying lindane alone to soil infested with Rhizoctonia solani.

Both the insecticides and the fungicides tested have an inhibitory effect on the growth of damping-off fungi, as demonstrated by the plate culture tests. With combinations of insecticides and fungicides in the medium, the total inhibitory effect was generally greater than the sum of the effects of the individual components. The test organism thus appeared to be more susceptible to either toxicant when simultaneously affected by the other toxicant.

The severity of pre-emergence damping-off has been shown by Leach (6) to vary inversely as the ratio between the velocity of seedling emergence and the growth rate of the pathogen. Inhibition of fungal growth can thus account for the improved emergence of seeds treated with insecticide in addition to a fungicide. It is possible for chlorinated hydrocarbon insecticides to affect the development of fungi in the soil at an appreciable distance from treated seeds, since both aqueous solutions and vapors of these compounds are fungitoxic, as shown by Richardson and Miller (10).

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JUNIPER HOSTS OF CEDAR-APPLE AND CEDAR-HAWTHORN RUST

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Abstract

All species, varieties, and forms of the genus Juniperus observed to be susceptible or resistant to cedar-apple rust, Gymnosporangium juniperi-virginianae, or cedar-hawthorn rust, G. globosum, are listed. This list is based on field observations made at the Morton Arboretum and on lists found in the literature. Two species, J. pinchotii and J. utahensis, and 13 varieties of other Juniperus species not found in previous reports are reported susceptible for the first time to cedar-apple rust. Nine additional varieties are reported susceptible to cedar-hawthorn rust.

INTRODUCTION

The injury caused by Gymnosporangium rusts is sufficiently severe in some years that these rusts are considered the most important diseases in commercial nursery and ornamental plantings. Cedar-apple rust, G. juniperi-virginianae Schw., and cedar-hawthorn rust, G. globosum Farl., are the most widespread and injurious rusts on junipers in Illinois. Although cedar-quince rust, G. clavipes Cke. & Pk., is present in Illinois, it usually is less conspicuous and causes less injury than the two previously mentioned rusts.

The control of rust diseases in apple orchards by the use of fungicide sprays and by the development of resistant varieties of apples has greatly reduced the necessity for destroying or removing the Juniperus host in the vicinity of orchards. As ornamental trees and shrubs, junipers have an integral part in present day landscaping designs. There is a definite need for a complete list of Juniperus species and varieties that are known to be susceptible or resistant to cedar-apple and cedar-hawthorn rusts. This list would serve nurserymen, arborists, and home owners as a guide for the selection of the more desirable varieties of junipers.

FIELD OBSERVATIONS

Observations on the rusts of junipers were made at the Morton Arboretum near Lisle, Illinois on May 22, 1959. Due to the proximity of Malus and Crataegus hosts and the compactness of the Juniperus collection (150 x 150 feet), all of the junipers were subject to a relatively uniform source of abundant natural inoculum of both cedar-apple and cedar-hawthorn rusts. Many of the susceptible varieties of junipers were so severely affected by rust that their ornamental value was destroyed. Because spore horn development by the rust galls was so pronounced in May of 1959, it was possible to determine the extent and severity of both rusts on the various species and forms of junipers. Data were obtained on the presence or absence of rust galls and the abundance of galls on each species, variety, and form of juniper growing in the Arboretum.

Each individual juniper in the Morton Arboretum is permanently tagged with its scientific name. The Morton Arboretum follows the classification of Rehder (11, 12) in naming the species, varieties, and forms in its juniper collection. However, a number of the juniper specimens in the Arboretum's collection have names which are not given in Rehder's listing or in any other plant listing examined. Rehder's classification is also followed in this publication. Where the forms or varieties were not listed by Rehder, Bailey's classification (2) is followed. Changes resulting from synonomy are incorporated in this list. The horticultural varieties listed without authorities are followed by the word Hort.

DISCUSSION

The susceptibility or resistance of the various species, varieties and forms of junipers in the Morton Arboretum collection are listed in Table 1. Also listed are the reports on susceptibility and resistance to cedar-apple and cedar-hawthorn rust given by other observers. Extensive lists of Juniperus varieties susceptible to cedar-apple rust were published by Crowell (6) and Boyce (4). In addition, several miscellaneous reports have been made on the susceptibility of junipers to cedar-hawthorn rust (1, 3, 5, 8, 9, 13).

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Table 1. Junipers susceptible and resistant to cedar-apple and cedar-hawthorn rusts.

Species, varieties, and forms of Juniperus	Cedar-apple rust		Cedar-hawthorn rust	
J. chinensis L.	R[a]	(R)[b]	R	
f. aureo-globosa Rehd.	R		R	
columnaris Hottes	R		R	
femina Bailey	R		R	
fortunei Hort.	R		R	
f. globosa (Hornibr.) Rehd.	S-2[c]	(R)[b]	R	
f. japonica (Carr.) Lipa	R	(R)[b]	R	
Keteleeri Cornman	R		R	
leeana	R		R	
f. mas (Gord.) Rehd.		(R)[b]		
var. oblonga	R		R	
f. pendula (Franch) Beiss.		(R)[b]		
f. Pfitzeriana (Spaeth) Rehd.	R	(R)[b] (S)[d]	R	
aurea	R		R	
f. plumosa (Hornibr.) Rehd.		(R)[b]		
var. plumosa aurea Hornibr.		(R)[b]		
f. pyramidalis (Carr.) Beiss.	R	(R)[b]	R	
var. Sargenti Henry	R	(R)[b]	R	
sylvestris plumosa Hort.	R		R	
f. variegata (Maxw.) Rehd.	R		R	
Watereri Hort.	R	(R)[b]	R	
J. communis L.		(R)[b] (S)[e]		(S)[f]
f. aurea (Carr.) Rehd.		(R)[b]		
f. aureo-spica (Rehd.) Lipa		(R)[b]		
f. compressa (Carr.) Rehd.		(R)[b]		
cracovica Carr.	R	(R)[b]	R	
var. depressa Pursh	R	(R)[b]	R	
var. hispanica Endl.	R		R	
var. oblonga Loud.		(R)[b]		
f. oblongo-pendula Beiss.		(R)[b]		
pyramidalis Hort.		(R)[b]		
var. saxatilis Pallas		(R)[b]		
f. stricta (Carr.) Rehd.	R	(R)[b]	R	
f. suecica (Mill.) Beiss.	R	(R)[b]	R	
nana Hort.	R		R	
J. conferta Parl.	R	(R)[b]	R	
J. davurica Pall.	R		R	
J. excelsa f. stricta (R.Sm.) Rehd.	R		R	
J. formosana Hayata	R	(R)[b]	R	
J. glauca hetzii	R		R	
J. glaucescens Florin	R		R	
J. hetzii	R		R	
J. horizontalis Moench	R	(S)[b]	R	(S)[g]
admirabilis	R		R	
adpressus	R		R	
f. alpina (Loud.) Rehd.	S-2	(R)[b]	R	
argenteus	R		R	
f. Douglasii Rehd.	R	(S)[b]	R	
eximius	R		R	
filicinus minimus	R		R	
f. glomerata Rehd.		(R)[b]		
hudsonica	R		R	
lividus	R		R	
petraeus	R		R	
f. plumosa Rehd.	R	(S)[b]	R	
compacta	R		R	
subglaucus	R		R	
f. variegata Slavin	R	(R)[b]	R	
Wiltoni	R		R	
J. japonica nana D. Hill	R		R	
J. macrocarpa Sibt. & Sm.	R		R	
J. monosperma (Engelm.) Sarg.	R		R	
J. Pinchotii Sudw.	S-1		R	
J. procumbens (Endl.) Sieb. & Zucc.	R	(R)[b]	R	
J. rigida Sieb. & Zucc.	R	(R)[b]	R	

Table 1. (concluded)

Species, varieties, and forms of Juniperus	Cedar-apple rust		Cedar-hawthorn rust	
J. Sabina L.	R	(R)[b]	R	
f. fastigiata (Beiss.) Voss	R		R	
pyramidalis Hort.		(R)[b]		
f. tamariscifolia (Ait.) Koeh.	R	(R)[b]	R	
f. variegata (West.) Beiss.		(R)[b]		
J. scopulorum Sarg.	S-2	(S)[b]		(S)[h]
funalis	S-2		R	
hilli D. Hill	S-2		R	
hilli argentea pyramidalis	S-3		R	
f. horizontalis (D. Hill) Rehd.	S-2	(R)[b]	S-3	
moffeti Hort.	S-2		R	
pendula D. Hill	S-3		R	
pillaris D. Hill	S-2		R	
f. viridifolia (D. Hill) Rehd.		(S)[b]		
J. silicicola (Small) Bailey		(S)[i]		(S)[i]
J. squamata Lamb		(R)[b]		
albo-variegata Hort.	R		R	
argesii	R		R	
var. Fargesii Rehd. & Wils.		(R)[b]		
var. Meyeri Rehd.	R	(R)[b]	R	
parsoni	R		R	
f. Wilsonii Rehd.		(R)[b]		
J. utahensis (Engelm.) Lemm.	S-3		R	
J. virginalis Hort.	R		R	
J. virginiana L.	S-3	(S)[j]	S-3	(S)[j]
aurea		(R)[b]		
Burkii Bailey		(R)[b]		
f. Canaertii (Senecl.) Rehd.	S-3	(R)[b]	S-3	
f. Chamberlaynii (Carr.) Beiss.	S-3	(S)[b]	S-1	
cinerascens Hort.	S-3	(S)[b]	S-3	
cupressifolia	R		R	
f. elegantissima (Hochst.) Rehd.	S-3	(S)[b]	S-3	
fastigiata Hort.		(S)[b]		
f. filifera (D. Hill) Rehd.		(S)[b]		
f. glauca (Carr.) Beiss.	S-3	(S)[b]	S-3	
f. globosa (Beiss.) Schn.	R	(S)[b]	R	
f. Kosteri (Beiss.) Lipa	R	(R)[b]	R	
horizontalis Arb. Kew.	R		R	
nova D. Hill	S-2		R	
f. pendula (Carr.) Beiss.	S-3	(S)[b]	S-2	
f. plumosa Schn.		(R)[b]		
polymorpha Hort.		(S)[b]		
pseudocupressus	R		R	
f. pyramidalis (Carr.) Beiss.	R	(S)[b]	R	
glauca		(R)[b]		
pyramidiformis D. Hill	S-3		S-3	
f. reptans (Beiss.) Rehd.	S-3	(S)[b]	R	
f. Schottii (Gord.) Beiss.	S-3	(S)[b]	R	
f. tripartita (Senecl.) Beiss.	R	(R)[b]	R	
f. variegata (Laws.) Rehd.	S-2		S-1	
f. venusta (Ellw. & Barry) Rehd.		(R)[b]		

[a] R-resistant.
[b] Reported as either resistant (R) or susceptible (S) by Crowell (6). All infection ratings that occur in parentheses are those by other authors.
[c] S-1, slight infection; S-2, moderate; S-3, severe.
[d] Reported susceptible by Ray (10).
[e] Reported susceptible by Kauffman (7).
[f] Reported susceptible in Pennsylvania by MacLachlan (8).
[g] Reported susceptible by Brenckle (5).
[h] Reported susceptible by Martin (9) and by Bliss (3).
[i] Reported susceptible by Stone (13).
[j] Reported susceptible to cedar-apple rust in 30 States and Ontario and to cedar-hawthorn rust in 21 States by Arthur (1).

Two species, J. pinchotii and J. utahensis, and 13 varieties and forms of other Juniperus species not found in reports on susceptible hosts to cedar-apple rust are reported here. Nine additional varieties not found in previous host lists are reported susceptible to cedar-hawthorn rust. Of the 116 species, varieties, and forms of junipers listed in Table 1, 36 are susceptible to cedar-apple rust and 14 are susceptible to cedar-hawthorn rust.

A careful selection of resistant species and varieties of junipers will reduce if not eliminate the problem of cedar-apple and cedar-hawthorn rusts. Particular attention should be given when selecting varieties of J. virginiana since 16 forms are susceptible to at least one or both rusts and 10 forms are believed resistant to both. J. scopulorum and all of its forms and varieties were observed to be susceptible to cedar-apple rust and should be used only if other juniper species are not available.

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REACTION OF CORN AND SORGHUM TO CORN AND SUDAN GRASS HEAD SMUTS[1]

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Summary

Two collections of corn head smut and one of Sudan grass head smut were hypodermically injected into seedlings of three varieties each of sweet corn and grain sorghum. Corn head smut infected only corn, but the Sudan grass head smut collection infected not only the varieties of grain sorghum but also one of sweet corn. The diseased sweet corn plants were much stunted and the tassels greatly proliferated. The results imply that head smutted Sudan grass may be a source of inoculum responsible for the appearance of head smut on some corn varieties.

INTRODUCTION

Sphacelotheca reiliana (Kuehn) Clint. induces head smut of corn (Zea mays), sorghum (Sorghum vulgare) and related grasses throughout the world. Reed, Swabey and Kolk (2) concluded from infested soil-cross inoculation trials that corn head smut was physiologically different from sorghum head smut. This conclusion has been accepted as stating the existence of a corn head smut race and a sorghum head smut race; Halisky, Smeltzer and Houston (1) recently failed to infect susceptible corn through soil by spores collected from smutted sorghum species. The distinction between the two races does not appear to be absolute, as Reed, Swabey and Kolk got head smut developing on a small percentage of corn and sorghum plants from the alternate smut. Sudan grass (Sorghum vulgare var. sudanense) head smut is generally held to be the same as sorghum head smut.

The purpose of the present work was to observe the reactions of corn and sorghum to collections of corn head smut by hypodermic inoculations and to note the reactions of these same plants to a collection of Sudan grass head smut.

MATERIALS AND METHODS

Two collections of corn head smut and one of Sudan grass head smut were used. The Sudan grass head smut from the variety Greenleaf and one of the corn head smut collections were obtained from Dr. P. M. Halisky of Davis, California, while the other corn head smut collection was obtained by the junior author from the State of Washington.

Mixed sporidia from four or five chlamydospores of each of the collections were grown on PDA and suspended in distilled water. The mixed sporidia were introduced hypodermically into 15 to 20 seedlings at the 3- to 4-leaf stage by injecting 1/2 ml of their suspensions in two locations on the stem to insure placing the inoculum at the growing point.

Table 1. Reaction of sweet corn and grain sorghum to three collections of head smut.

Variety	Smut collection		
	California		Washington
	Corn	Sudan grass	Corn
Sweet Corn:			
Golden Beauty	+	-	+
Sugar & Gold	+	-	+
North Star	+	+	+
Grain Sorghum:			
Reliance	-	+	-
Dual	-	+	-
Norghum	-	+	-

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FIGURE 1. North Star sweet corn plants. Left -- healthy. Right -- stunted from Sudan grass head smut inoculation.

FIGURE 2. North Star sweet corn plants inoculated with Sudan grass head smut, showing leafy tassels and stunted stalks.

EXPERIMENTAL RESULTS

Three varieties each of sweet corn and grain sorghum grown in the greenhouse were injected with two collections of corn head smut and one collection of Sudan grass head smut. The results are shown in Table 1.

Corn head smut developed on corn and not on sorghum as generally accepted. Sudan grass head smut developed on all three varieties of sorghum and on one variety of sweet corn, North Star. The susceptibility of North Star sweet corn to this head smut was confirmed in a repeat trial.

Mixed sporidial cultures from chlamydospores taken from North Star sweet corn infected with Sudan grass head smut were reinfective to North Star sweet corn and to Sugar Drip sorghum. This implies the possibility of obtaining head smut on corn from inoculum on plants other than corn.

The North Star sweet corn plants infected with Sudan grass head smut were stunted (Fig. 1); their tassels were leafy structures in many instances (Fig. 2); and their ears either failed to form or they were small, with some kernels replaced by smut spores.

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SOUTH DAKOTA AGRICULTURAL EXPERIMENT STATION, BROOKINGS

SOUTHERN BLIGHT OF BIRDSFOOT TREFOIL (LOTUS CORNICULATUS)[1]

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Summary

The southern blight fungus (Sclerotium rolfsii Sacc.) attacked birdsfoot trefoil (Lotus corniculatus) at Holland, Virginia in 1958 and 1959. This disease organism has not previously been reported as a pathogen of L. corniculatus. It attacks and girdles plants near the soil line, causing rapid death of aboveground parts. The foliar symptoms of southern blight resemble those of plants girdled by Rhizoctonia solani Kuehn. However, S. rolfsii is less pathogenic to birdsfoot trefoil than R. solani. Pathogenicity tests suggest that S. rolfsii is most effective as a pathogen to birdsfoot trefoil when a source of organic matter is present on which to build-up its initial growth before initiating infection.

INTRODUCTION

Southern blight (Sclerotium rolfsii Sacc.) was found on birdsfoot trefoil (Lotus corniculatus) at Holland, Virginia during the early part of September 1958 and during July, August, and September 1959. The southern blight fungus was previously reported attacking L. uliginosus (3) but not L. corniculatus.

SYMPTOMS

The southern blight organism attacks plants at or near the soil line, producing grayish-tan lesions that enlarge and girdle the stem. Infected plants are usually wilted within 3 to 4 days. Leaves of diseased plants first turn grayish-green then cinnamon-brown, resembling foliage of plants mechanically girdled. During warm, rainy weather white, cottony mycelium of the fungus frequently covers the basal stem parts of infected plants (Fig. 1). The fungus may attack only one plant in an area or it may spread to several adjacent plants. In spaced plants where many shoots develop from a crown, the fungus sometimes spreads progressively from a few diseased shoots to the entire plant.

Sometimes sclerotia of the fungus were found on infected plants. When moisture was not limiting, small, white sclerotia that turned orange to brown with age, characteristic of S. rolfsii, developed on diseased stems (Fig. 1). Diseased tissue from the margin of stem lesions and sclerotia when plated on potato-dextrose agar yielded cultures of S. rolfsii.

The foliar blight and stem girdling symptoms caused by Rhizoctonia solani sometimes resemble symptoms resulting from infection by S. rolfsii. These two diseases can be differentiated, however, because R. solani usually attacks birdsfoot trefoil stems 1/2 to 1 inch or more above the soil line, producing light-tan lesions bordered by narrow, convex, irregular brown bands, whereas S. rolfsii attacks stems at the soil line and no brown bands border the lesions.

PATHOGENICITY TEST

During the spring of 1959 greenhouse inoculation tests were conducted with isolates of S. rolfsii to study pathogenicity and disease development. Three groups of plants, each consisting of 44 two-month-old rooted birdsfoot trefoil cuttings, were exposed to different kinds of inoculum. In one group dried grain inoculum was placed around the base of each plant (2). In a second group a macerated culture of the fungus mycelium was sprayed over the plants. The third group was inoculated by pouring a suspension of fungus sclerotia around the base of the plants. Twenty check plants were sprayed with distilled water. Inoculated and check plants were placed in a greenhouse moist chamber for 144 hours at 80° F.

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[1]Cooperative investigation between the Crops Research Division, Agricultural Research Service, United States Department of Agriculture, and the Virginia Agricultural Experiment Station. Published with the approval of the Director of the Virginia Agricultural Experiment Station.

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FIGURE 1. Birdsfoot trefoil stems from a plant killed by S. rolfsii. The stem on the left is covered, at a branch, by heavy aerial mycelium of the fungus; the other two stems have sclerotia of the fungus attached.

FIGURE 2. Birdsfoot trefoil plants grown from rooted cuttings. At right, healthy check plant; at left, plant killed by S. rolfsii showing mostly stem and branch tissue with a few dry and shriveled leaves.

RESULTS AND CONCLUSIONS

Greatest infection occurred in plants inoculated with dried grain inoculum. The percentage of plants infected by use of the various types of inoculum varied as follows: grain inoculum, 87 percent; fungus mycelium, 23 percent; fungus sclerotia, 20 percent. No infection occurred in check plants. The contrast between diseased and healthy plants is shown in Figure 2.

The results suggest that S. rolfsii increases on organic matter before initiating infection. These results confirm those reported by Garren (1) who found that S. rolfsii caused more infection on peanuts growing in soil with a surface mulch than when the cover crop, debris, or surface organic matter was buried.

In general S. rolfsii is much less pathogenic than R. solani to birdsfoot trefoil. Only minor disease problems are anticipated with S. rolfsii except in certain localized areas where it is known to be a severe pathogen of other crops.

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OBSERVATIONS ON THE PASSAGE OF PEACH NECROTIC LEAF SPOT AND PEACH RING SPOT VIRUSES THROUGH PEACH AND NECTARINE SEEDS AND THEIR EFFECTS ON THE RESULTING SEEDLINGS[1]

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Summary

Observations based on seedling expression or on index plant reactions suggest seed transmission of both peach ring spot and peach necrotic leaf spot viruses. Seed transmission of peach ring spot virus appeared to occur in from 1.1 to 11.7 percent of the seeds in four lots. Seed transmission of peach necrotic leaf spot virus apparently occurred in from 3.0 to 9.0 percent of the seeds in three lots. The evidence indicates that all seedlings infected with peach necrotic leaf spot virus also were carrying peach ring spot virus. They gave a reaction on Shiro-fugen flowering cherry which could not be distinguished from that normally produced by peach ring spot virus. Measurements of the heights of 149 seedlings of one population showed a significant difference in favor of those that tested negative over those that tested positive on Shiro-fugen.

Subsequent to the report by Wagnon and Breece (5) in 1954 on the prevalence of peach necrotic leaf spot virus (PNLSV) in California, this virus, like that causing peach ring spot (PRSV), has been of considerable concern in the selection of peach and nectarine propagating material. In peach, leaf symptoms commonly develop during the first year after infection with PRSV and then the tree usually becomes symptomless, but the symptoms of PNLSV may recur in peach each year subsequent to infection. Detection of PNLSV has been by visual inspections of peach and nectarine trees during specific periods of summer growth, whereas PRSV commonly has been detected by index tests on various hosts. The symptoms of PNLSV are particularly prominent on succulent growth of young rapidly growing trees in the nursery. In California, where June-budding is commonly practiced, the forcing of buds on peach rootstock brings about succulent growth during periods of warm summer temperatures. June-budded peach trees therefore provide an ideal medium for detection of this virus. Symptoms may be present on both the rootstock sprouts and the shoot developing from the variety bud. Since mature trees which usually serve as budwood sources do not consistently show symptoms of PNLSV when infected, budwood sources free of this virus were sought by checking the progeny in the nursery row (6). Work carried on during 1955-57 indicated that a high percent of June-budded progeny of an infected budwood source tree would show symptoms when examined at the proper time. On some occasions the June-budded progeny of specific budwood source trees thought to be free of PNLSV, and which tested negative on Shiro-fugen, showed a low incidence (1/2 to 1 percent) of PNLSV. The progeny was propagated on commercial Lovell seedling rootstocks and it was thought that this low percent of infected progeny might be attributed either to the passage of the virus through the Lovell seed or to natural spread by a vector. An opportunity to obtain information relative to both possibilities came about incidental to other work carried on during 1958 and 1959.

MATERIALS AND METHODS

Information on the Lovell seedlings was obtained from 130 such seedlings grown in Placer County, California, from commercial seed planted in the spring of 1958. These seedlings were purchased from a nurseryman for use as rootstock in a "virus free" test plot. As it was desirable to have trees free of viruses for planting in the test plot, each seedling growing in the nursery row received two visual inspections and a PRSV index test on Shiro-fugen as a

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[1]Part of the information in this paper was obtained through work done under Agricultural Marketing Service Project number SDA-Calif. -A-2, for which State funds were matched with federal funds received from the Agricultural Marketing Service, United States Department of Agriculture, under provisions of the Agricultural Marketing Act of 1946.

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check for viruses; only those found free were used in the "virus-free" test plot.

Additional data were obtained from 1227 seedlings which were part of a peach and nectarine breeding program being conducted at the U. S. Horticultural Field Station, Fresno, in Fresno County, California. These seedlings, which for the most part were the results of hand-pollinated crosses made in 1957, were the progeny of three trees of Alamar peach, one tree each of Fay Elberta, Redglobe, F-11 and FV 234-1 peach, and one tree of Le Grand nectarine. All were from seeds germinated during the winter of 1957-58 in flats in a greenhouse and then transferred in the spring of 1958 to the field, where they were planted at 4-foot intervals in rows approximately 12 feet apart.

RESULTS

In this paper, reference to a plant as infected with PNLSV is based upon the presence of visual leaf symptoms of the disease, and reference to a plant as infected with PRSV is based upon a positive reaction on Shiro-fugen flowering cherry (2, 3).

The earliest visual inspections, which were made in mid July 1958, proved to be too early. Only about half of the total cases of PNLSV noted in September and October had shown symptoms in mid July. No symptoms recognizable as being caused by PRSV were observed on any of the seedlings during the summer of 1958 or early spring of 1959. This was expected even though the seedlings were infected with PRSV, as Cochran (1) reported that peach seedlings grown from seed infected with PRSV are symptomless. All of the seedlings exhibiting symptoms of PNLSV, as well as some others which showed no visual symptoms of any virus, gave a positive reaction on Shiro-fugen. No difference in the Shiro-fugen reactions was noted between seedlings showing symptoms of PNLSV and those which did not show symptoms of any virus but which gave a positive reaction indicative of PRSV.

Seed transmission rather than spread resulting from vectors is believed to account for the occurrence of all the cases of PNLSV and PRSV (Table 1). This is indicated particularly by the seedlings grown in Fresno County. The seedlings of FV 234-1, Fay Elberta and Alamar were found to be 1.1 to 11.7 percent infected with PRSV. Those of Fay Elberta and Alamar were 3.5 and 9 percent infected with PNLSV (the seedlings of FV 234-1 were not inspected for PNLSV). The progeny of Redglobe, Le Grand and F-11, which indexed negative on Shiro-fugen and showed no evidence of virus infection, tended to substantiate the absence of natural spread among the progenies. The passage of PNLSV through Lovell seed can account for the low percent of cases previously mentioned in June-budded nursery stock propagated on Lovell seedling rootstocks.

Table 1. Results of visual inspections and Shiro-fugen tests of peach and nectarine seedlings.

Seed Source		Number of	Infected seedling		Percent infected	
Variety	No. of trees	Progeny Grown	PNLSV	PRSV	PNLSV	PRSV
Lovell	Unknown	130	4	11	3.0	8.4
Alamar	3	375	34	44	9.0	11.7
Redglobe	1	192	0	0	.0	.0
Le Grand (nectarine)	1	10	0	0	.0	.0
F-11	1	128	0	0	.0	.0
Fay Elberta	1	339	12	14	3.5	4.1
FV 234-1	1	183	--[a]	2	--[a]	1.1

[a]No visual inspections were made of these seedlings.

Measurements of heights of 149 seedlings representing one of the populations, Alamar x Dixired, were made in November 1958, after one season's growth. One-hundred six seedlings which showed no evidence of virus infection by visual inspections and the Shiro-fugen tests averaged 54.77 inches in height, whereas 43 seedlings which gave a positive test on Shiro-fugen averaged 46.58 inches in height. The difference in height of 8.19 inches was highly significant, as only .04 inch was necessary for significance at the .05 level.

DISCUSSION

The passage of PRSV through peach seed has been reported previously by Cochran (1) and Millikan (4); but seed passage of PNLSV has not been reported previously. Since the 130 Lovell seedlings in the present study probably were the progeny of more than one tree, the percent of

PNLSV and PRSV seed transmission is not indicative of the total number that might be expected had they all originated from one infected tree. However, the percentage is indicative of the amount of seed transmission that may occur in one commercial source of Lovell seed. Like that from the Lovell seed, the data from the Alamar seed were likewise from more than one tree; however, it is of interest that of the 375 seedlings of three Alamar trees 11.7 percent were infected as compared with 1.1 percent infected of the 183 progeny from one FV 234-1 tree. The PRSV index tests on Shiro-fugen were made on several of the parent trees, but in some instances the parent trees were removed subsequent to the harvesting of the seed and before data relative to their virus content could be obtained. One of the three Alamar trees, the Redglobe tree and the F-11 tree gave a negative test on Shiro-fugen, whereas the Le Grand nectarine and FV 234-1 trees both tested positive.

Various peach and nectarine source trees including Cardinal, Rio Oso Gem, F-14, 67-43, F-15, Freedom, Merrill Fiesta, and Dixired, served as pollen parents for the progeny of Alamar (a sterile-pollen variety), Redglobe, Le Grand, F-11 and Fay Elberta varieties. The FV 234-1 and Lovell progeny were the result of open pollination. These two varieties have fertile pollen, therefore it is assumed that probably 90 percent of their progeny are the result of self fertilization.

Unfortunately, complete data on the virus content of the various pollen parents are unavailable, nor were records maintained so as to identify the pollen parent tree of a specific lot of seedlings.

The data indicate that whenever PNLSV was seed transmitted PRSV was transmitted simultaneously, and that PRSV was transmitted singly in a minority of cases. Since PNLSV symptoms may not occur on trees which are not actively growing during high summer temperatures, further observations of these trees or their progenies would be necessary to give assurance that PNLSV is not present in all cases where PRSV was found to occur singly.

On the basis of previous work reported by Cochran (1) and Millikan (4), PRSV would be expected to occur in a small percent of the progeny of the single Le Grand tree which tested positive on Shiro-fugen. The complete absence of PRSV in any of these progeny might be attributed to the fact that the population tested was too small or that PRSV is not seed-borne in all varieties or all trees.

That vigor and growth of seedlings was adversely affected by virus carried through the seed was indicated in the growth comparison of healthy and affected seedlings. Seedlings giving a positive Shiro-fugen test were approximately 15 percent smaller than those having a negative test. This reduction in growth by virus is important to nurserymen, where size of nursery tree determines the price. Whether orchard trees grown or propagated from infected trees are similarly affected remains to be proved.

It is not known whether the amount of seed passage of either PNLSV or PRSV, as indicated in seedlings, is affected by such factors as: 1) variation in strains of the two viruses; 2) variety of peach and nectarine used as seed- and pollen-parent, and 3) whether a flower is cross- or self-fertilized. Further tests are in progress to obtain additional information on possible factors involved:

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ALFALFA MOSAIC VIRUS IN WHITE CLOVER AND POTATOES[1]

P. M. Halisky, Byron R. Houston, and A. R. Magie

Summary

Two strains of alfalfa mosaic virus (AMV) were studied and their host range, symptomatology, physical properties, and cross-protection relationships determined. Based on combined criteria the two strains were identified as alfalfa mosaic virus N and common alfalfa mosaic virus respectively. Potato was found to be a good indicator plant for differentiating between strains of AMV. The differential response of four potato varieties is described and illustrated. Both strains of AMV were found to be tuber transmitted (28.2 and 41.3 percent) although tuber necrosis was not evident.

More than 100 viruses infecting legumes have been described in the literature (19). Among the legume viruses commonly occurring in white clover (Trifolium repens) are many strains of alfalfa mosaic virus (AMV). At least eight distinct strains of this virus have been described as follows: alfalfa mosaic virus N (7), alfalfa yellow mosaic (24), common alfalfa mosaic (14, 17, 23), bean yellow dot (21), Ladino clover yellow patch (6), pepper mosaic (1), potato calico (2, 12, 15), and potato tuber necrosis (12). Most of these strains are known to be infectious to white clover. In California AMV has been reported occurring naturally in crops such as alfalfa (22), celery (20), Ladino white clover (3), and potatoes (12, 15, 16). Two closely related strains of AMV were selected from among 17 collections of infected white clover for detailed study, characterization, and identity. In this study these two strains were tentatively designated as AMV-3 and AMV-7 respectively.

HOST RANGE AND SYMPTOMATOLOGY

Host-range studies were conducted with 50 species of leguminous and non-leguminous plants using sap inoculation in regularly fumigated greenhouses. Repeated studies indicated that 38 plant hosts shown in Table 1 were infected by either one or both strains of AMV while 12 other species not listed in the table were either resistant or immune to infection. The infection data shown in Table 1 indicate that the greatest majority of host plants were systemically infected by both virus strains. Among these the most severe systemic infection, stunting, and necrosis were observed in 14 species of clovers. Only one species of clover, Trifolium fragiferum (strawberry clover), was observed to be resistant to infection. Host plants which consistently responded with local-lesion reactions to both strains of AMV were globe amaranth, colored pigweed, sweet pea, baby lima bean, Bountiful bean, and blackeye cowpea. Other hosts which developed local lesions initially but later expressed systemic necrosis as well were broad bean, soybean, fenugreek, potato, and tobacco. Several crop plants such as beets, castor beans, spinach, sugar pumpkin, sunflower, and watermelon were found to be refractory to systemic infection. Initially these plants developed a few small, chlorotic, local lesions in inoculated leaves but later recovered from inoculation shock and showed no further symptoms. Other plants such as guar, lupines, trefoil, kudzu, and sweetpotato were apparently immune to infection by both strains of AMV studied here.

In general, host-range studies alone are inadequate as criteria in defining virus strains. Different varieties of tomatoes and cowpeas inoculated with numerous strains of AMV showed great variability in susceptibility and symptomatology according to Milbrath and McWhorter (9, 10). Such variations indicate the inherent dangers in host-range studies. Furthermore, the occurrence of mixtures of legume viruses in field collections (3, 11, 13, 19, 23) is another factor which detracts greatly from the validity of host-range studies. Identity of virus strains must therefore be based on a combination of criteria including physical properties, cross-protection studies, differential symptomatology, and host-range studies.

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[1] Contribution from the Agricultural Experiment Station, Department of Plant Pathology, University of California, Davis.

Table 1. Reaction of two strains of alfalfa mosaic virus on selected leguminous and non-leguminous host plants.

Host	Common name or variety	Strain of AMV and reaction type[a]	
		AMV-3	AMV-7
Leguminous species			
Cicer arietinum	Garbanzo	S	S
Glycine max	Soybean	LL, S	LL, S
Lathyrus odoratus	Sweet pea	LL	LL
Medicago hispida[b]	Bur clover	S	S
Medicago lupulina[b]	Hop clover	-	S
Medicago sativa[b]	Alfalfa	S	S
Melilotus alba[b]	White sweetclover	S	S
Melilotus alba annua[b]	Hubam white clover	S	S
Melilotus indica[b]	Sour clover	S	S
Melilotus officinalis[b]	Yellow sweetclover	S	S
Phaseolus lunatus	Baby lima bean	LL	LL
Phaseolus multiflorus	Scarlet runner bean	S	S
Phaseolus vulgaris	Bountiful bean	LL	LL
Pisum sativum	Field pea	S	S
Trifolium alexandrinum[b]	Berseem clover	S	S
Trifolium hirtum[b]	Rose clover	S	S
Trifolium hybridum[b]	Alsike clover	S	S
Trifolium incarnatum[b]	Crimson clover	S	S
Trifolium pratense[b]	Red clover	S	S
Trifolium repens[b]	Ladino white clover	S	S
Trifolium subterraneum[b]	Subterranean clover	S	S
Trigonella foenum-graecum	Fenugreek	LL, S	LL, S
Vicia faba	Broad bean	LL, S	LL, S
Vigna sinensis	Blackeye cowpea	LL	LL
Non-leguminous species			
Apium graveolens	Celery	S	S
Capsicum annuum	Pepper	S	S
Chenopodium amaranticolor	Pigweed	LL	LL
Cucumis melo	Persian melon	S	S
Cucumis sativus	Cucumber	S	S
Datura stramonium	Jimson weed	S	S
Gomphrena globosa	Globe amaranth	LL	LL
Hibiscus esculentus	Okra (Gumbo)	S	S
Lycopersicon esculentum	Tomato	LL	LL
Nicotiana glutinosa	Tobacco	LL, S	S
Nicotiana rustica	Tobacco	S	S
Nicotiana tabacum	Turkish tobacco	LL, S	S
Solanum tuberosum	Potato	LL, S	S
Zinnia elegans	Zinnia	S	S

[a] The reaction types recorded for each species are local lesion (LL), systemic infection (S), both reactions (LL, S), or none (-).

[b] Severe systemic infection, stunting, vascular necrosis, and premature death were often produced by both strains of virus in 14 species of clovers (Medicago, Melilotus, and Trifolium).

PHYSICAL PROPERTIES AND CROSS-PROTECTION

Determinations of the physical properties of strains AMV-3 and AMV-7 were made to substantiate their identity. These determinations were conducted during two seasons and a summary of the data is presented in Table 2. Variations in consecutive determination of physical properties were observed occasionally and attributed to differences in virus concentration. AMV does not have a sharp thermal inactivation point according to Milbrath (8) but extends over a range of 20° C or more. Infective particles survive directly in proportion to

Table 2. Comparison of physical properties and identity of two strains of alfalfa mosaic virus isolated from Ladino white clover.

Physical property[a]	Strain of alfalfa mosaic virus AMV-3	AMV-7
Aging in vitro at 21° C	48 hours	72 hours
Dilution end point	1:2000	1:5000
Thermal inactivation point	50° - 55° C	61° - 64° C
Strain identity	strain of alfalfa mosaic virus N (7)	strain of common alfalfa mosaic virus (14, 23)

[a] Blackeye cowpea (Vigna sinensis) was used as the local lesion indicator host for checking the physical properties of these strains.

the virus concentration in the original sap which may produce discrepancies in successive determinations of the T.I.P. for AMV. Based on host range, symptomatology, and physical properties the two strains studied here were found to closely resemble alfalfa mosaic virus N (7) and common alfalfa mosaic virus (14, 23) respectively.

Cross-protection studies were also conducted to determine the relationships of the two strains to a systemic strain of AMV and a systemic strain of cucumber mosaic virus (CMV). Blackeye cowpeas were used as the local lesion host for these studies. Cross-protection is based on the assumption that avirulent strains of a virus protect plants from the effect of virulent strains of the same or closely related viruses. Protection by the first virus was indicated when the second or challenge virus failed to produce symptoms. Results of studies showed that 244 cowpea plants infected with systemic AMV were refractory to subsequent infection with strains AMV-3 or AMV-7. The inhibition of necrotic local lesion formation by these strains in cowpeas is additional evidence of their identity as strains of AMV. Similar studies were conducted with CMV using systemic CMV as the initial virus and the two AMV strains as challenge viruses. Studies with 42 cowpea plants inoculated with systemic CMV showed no cross protection against subsequent infection by AMV-3 or AMV-7, both viruses producing prominent local lesions. Differentiation of AMV strains from CMV was also indicated by their positive reactions in Table 1 on Bountiful bean, broad bean and cowpea, using criteria established by Milbrath and Young (11). These workers found that both viruses may occur as components of a naturally-occurring mixture in Daphne odora.

DIFFERENTIAL RESPONSE OF POTATO VARIETIES

According to the literature (12, 13, 15, 16) alfalfa mosaic virus is known to produce a great variety of symptoms in potato ranging from calico to severe stem and tuber necrosis. However, the use of the potato as an indicator plant for segregating strains of AMV into distinct groups merits consideration. Host-range studies with strains of AMV led to the observation that some strains produce primarily necrotic symptoms in potato while others produce pronounced yellow mottling. A study was made of the differential characteristics induced by strains AMV-3 and AMV-7 in Russet Burbank (Netted Gem), White Rose, Kennebec, and Pontiac varieties of potato. Since the reaction to each virus was generally similar in all four varieties a summary of the differential symptoms was compiled (Table 3). In general the necrotic strain (AMV-3) identified as alfalfa mosaic virus N produced black, necrotic lesions on the leaves (Fig. 1A) and systemic necrosis in veins, petioles, and vines. In contrast to these symptoms the response of potatoes to common alfalfa mosaic virus (AMV-7) was characterized by pronounced systemic yellowing, rugosity, and spindliness (Fig. 1B, 1C).

TUBER TRANSMISSION OF AMV STRAINS

Tubers of certified potato varieties were planted in sterile soil in the greenhouse and the young plants subsequently inoculated with strains AMV-3 and AMV-7. All inoculated plants developed conspicuous foliage and vine symptoms (Fig. 1, Table 3). Tubers from both infected vines and healthy control plants were sliced and examined for internal necrosis. None of the tubers examined during a 2-year period of study showed any phloem necrosis or discoloration. When sliced tubers (seed pieces) were replanted considerable virus transmission was

Table 3. Differential characteristics induced by two strains of alfalfa mosaic virus in potatoes[a].

Host response	Strain of alfalfa mosaic virus	
	AMV-3 (necrotic)	AMV-7 (common)
Dwarfing	slight	absent
Infectivity	moderate	pronounced systemic infection
Spindliness	absent	vines spindly and distorted
Foliage yellowing	calico blotching	pronounced systemic yellowing
Foliage distortion	absent	severe rugosity and puckering
Leaflet necrosis	necrotic local lesions	absent
Stem necrosis	black necrotic streaks on vines and petioles	absent
Tuber necrosis	absent	absent
Tuber transmission	positive	positive

[a] The differential symptoms produced were similar in the four potato varieties used, namely Russet Burbank (Netted Gem), White Rose, Kennebec, and Pontiac.

FIGURE 1. Primary symptoms of alfalfa mosaic virus strains in potatoes. A -- Russet Burbank potato infected with AMV-3 (alfalfa mosaic virus N) showing necrotic local lesions. B -- Russet Burbank potato infected with AMV-7 (common alfalfa mosaic virus) showing foliage rugosity and vine spindliness. C -- White Rose potato infected with AMV-7 showing a systemic mottle.

observed in the young plants. The extent of tuber perpetuation of two strains of AMV in four potato varieties is shown by the data in Table 4. These data indicate that both strains of AMV were readily tuber-perpetuated even though tuber necrosis was not evident. The variety White Rose apparently did not transmit AMV-3 through tubers. Attempts to recover the virus from seedlings of this variety failed, ruling out the possibility of a symptomless carrier as was reported for the lucerne mosaic virus in potato (18).

DISCUSSION

Strains of AMV commonly occur in white clover under field conditions. The consequences of systemic infection in clover are weakened, less vigorous plants predisposed to drought damage and winter injury (3, 4, 5). AMV infection in clovers also results in substantial reduction in both seed and forage yields. Studies in the greenhouse indicated that strain AMV-3

Table 4. Tuber transmission of two strains of alfalfa mosaic virus in four potato varieties[a].

Potato variety	Strain of alfalfa mosaic virus		
	AMV-3	AMV-7	Checks
Russet Burbank			
Total plants	47	53	21
Virus-infected plants	20	16	1
Percent transmission	42.6	30.2	4.8
White Rose			
Total plants	38	39	30
Virus-infected plants	0	16	0
Percent transmission	0[b]	41	0
Kennebec			
Total plants	16	11	21
Virus-infected plants	12	8	0
Percent transmission	75	72.7	0
Pontiac			
Total plants	23	18	18
Virus-infected plants	3	10	0
Percent transmission	13	55.6	0
Average percent transmission	28.2	41.3	1.1

[a] Tubers of certified potato varieties were obtained from Dr. Herman Timm, Department of Vegetable Crops, University of California, Davis.
[b] Attempts to recover the virus from symptomless White Rose seedlings failed, indicating that AMV-3 was probably not completely systemic in this variety.

reduced the number of flowering heads in infected clones of red clover by 29 percent, in white clover by 41 percent, and in alsike clover by 87 percent respectively. Not only was the number of flower heads reduced greatly but the number of seeds in the heads of infected clover was much lower.

According to the literature, strains of AMV occur naturally in crop plants such as alfalfa (7, 22), beans (14, 21, 24), celery (20), peas (7, 23), pepper (1), potato (12, 13, 15, 16, 18), Ladino white clover (3, 6) as well as in ornamentals and leguminous weeds. AMV strains surviving in perennial hosts during winter months are carried by aphid vectors to field, forage, and vegetable crops during the growing season. A succession of different crop plants may thus comprise an infection chain for perpetuating and increasing strains of AMV. In California, for example, the occurrence of AMV in potato is invariably associated with adjacent alfalfa or white clover plantings (12, 13). In addition to the existence of a complex intercrop infection chain, studies dealing with AMV strains are further complicated by variability in symptomatology and susceptibility in different varieties within a species (9, 10, 13, 18), by differences in virus titer or concentration in extracted juice (8), and most important by the existence of strains as components of naturally-occurring virus mixtures in host plants under field conditions (3, 11, 13, 19, 23).

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HETEROGENEITY IN THE NORKA DIFFERENTIAL WHEAT VARIETY TO A NEW RACE OF ERYSIPHE GRAMINIS TRITICI

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Summary

The pathogenic reaction of a new race of powdery mildew of wheat (Erysiphe graminis f. sp. tritici (Em. Marchal)) in California is described. The new race separated the differential wheat variety Norka into two morphologically indistinguishable types -- one resistant and one susceptible. This "Norka" race is different from all other described races in its reaction on Norka wheat itself. Aside from its reaction on Norka it is different from all other races except race 3 and Arg. 3.

In a genetic study of powdery mildew resistance in wheat, a collection of mildew was used which seemed different from any previously reported. It was collected in the fall of 1950 in the summer experimental nursery at Davis, California. Perpetuated on White Federation wheat seedlings in the laboratory or greenhouse for 3 years, it remained stable in several tests on a series of varieties.

All tests for mildew reaction were conducted in the greenhouse at Davis during the winter. When conditions permitted thermostatic control, temperatures were maintained at about 17° to 18° C; at other times the maximum day temperature reached 30° or more.

In some tests inoculations were made at two stages of growth -- the one-leaf and three-leaf stages. In other tests, plants were inoculated only in the three-leaf stage. Both the leaf blade and leaf sheath were observed for mildew reaction. Readings were made 11 to 13 days after inoculation.

RESULTS

Table 1 shows the reaction of several varieties of wheat to the mildew collection under study, called "Norka" race. This race appeared different from all races previously reported by other workers (2, 3, 4, 5, 6, 7, 8) in its reaction on Norka wheat itself. Both resistant and susceptible plants were observed. A sample of the original Norka seed stock gave 31 resistant and 8 susceptible plants. Progeny tests in the greenhouse for mildew reaction of 17 other individual Norka plants morphologically identical at maturity gave 14 homozygous resistant and 3 homozygous susceptible lines. Progenies in the field of 4 of the 17 plants, 2 from each infection type, gave plants that were identical in all observed characters. It thus appears that the Norka variety itself consists of two homozygous strains for reaction to this mildew collection, and that the two infection types were not the result of mechanical seed mixing or recent natural outcrossing. The resistant and susceptible types were respectively designated Norka-1 and Norka-2.

Even aside from the reaction on Norka wheat, this race is certainly different from all other described races in the literature (2, 3, 4, 5, 6, 8, 9) except races 3 and Arg. 3 reported by Newton and Cherewick (3) and Vallega and Cenoz (9), respectively. The six races reported by Schlichting (7) were not considered since Chul was the only variety in common besides Norka. The pathogenic reaction of races 3 and Arg. 3 was reported respectively on only 3 and 6 varieties tested in common.

Leaf-blade reactions in the one-leaf and three-leaf stages to the Norka race were essentially identical for nearly all varieties (Table 1). For three varieties tested, however, resistance was higher in the three-leaf than in the one-leaf stage. Contrast was greatest with Michigan Amber Selection and Sturgeon, which were nearly fully susceptible in the seedling stage but immune in the three-leaf stage. The same was true for Hardired 5, though the difference was not so great. No varieties exhibited the reverse behavior.

The infection type of the leaf sheath corresponded very closely to that of the leaf blade in both stages of growth for most varieties. One striking exception was Michigan Amber Selection. When inoculated in the three-leaf stage, the leaf blades exhibited an immune reaction but the leaf sheaths, especially that of the first leaf, showed very heavy coverage and abundant sporulation. Neither Sturgeon nor Hardired 5 reacted like Michigan Amber Selection.

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Table 1. Powdery mildew reaction on the leaf blade of wheat varieties in the one-leaf and three-leaf stages of growth to the Norka race.

Variety[a]	R.L. No.	C.I. No.	Norka race: 1-leaf stage	Norka race: 3-leaf stage
Axminster[d]	75	8195	0_1[b]	0_1
Chul[d]	543	2227	4	4
Converse		4141	0_1	0_1
Dixon		6049	4	4
Hardired 5		12411	3	0_1, 1
Hope[d]		8178	0_1, 1, 2	0_1, 1
Hope x Hussar		11682	1	0_1, 1
Huron[d]		3315	0_1	0_1
Huron[d]	20	12663	4	4
T. timopheevi x (Illinois No. 1 x Chinese)[2]		12633	0_2	0_1
Indian		4489	4	3+
Leap		4823	4	4
Malakoff		4898	4	4
Michigan Amber Selection		11770	4[c]	0_1
Norka-1[d]	1888	4377	0_1	0_1
Norka-2[d]	1888	4377	4	4
Normandie[d]		12747	0_1	0_1
Purplestraw		1915	4	4
Sonora		3036	3	3
Sturgeon		11703	3[c]	0_1
Suwon 92[d]		12666	4	3+, 4
Trumbull[2]-Red Wonder-(Steinweidel-Timopheevi)		12559	1	0_1, 1
Ulka[d]		11478	0_2	0_1, 0_2

[a]Thanks are due to the late Dr. C. V. Lowther, U.S.D.A., Beltsville, Maryland, for providing the seed.

[b]Explanation of symbols: 0_1, immune, no visible signs of infection; 0_2, highly resistant, chlorotic or necrotic flecks present with no visible development of mycelium; 1, very resistant, slight development of mycelium but with little or no sporulation; 2, moderately resistant, moderate development of mycelium with slight production of conidia; 3, moderately susceptible, moderate to abundant development of mycelium accompanied by moderate sporulation; 4, very susceptible, abundant mycelium with large pustules abundantly sporulating, no chlorosis or necrosis. The use of (+) and (-) signs indicates a quantitative increase or decrease in the infection type. (Modified from Newton and Cherewick (3)).

[c]Not heavy coverage, but pustules sporulating very abundantly.

[d]Varieties grown as a test series during the 1950-1953 period.

DISCUSSION

The occurrence of two different infection types within the Norka wheat variety has not been reported by any previous worker, though the same seed stock presumably has been used by all other workers. Assuming that is so, three possible hypotheses remain why others have not observed the two infection types or a non-uniform reaction within the Norka variety itself. First, other workers may not have grown large enough populations to observe the less frequent susceptible type. In this hypothesis and the following one it is assumed that Norka-1 is genotypically Ml_tMl_t, and Norka-2 ml_tml_t. Pugsley and Carter (5) showed that Norka carries only the Ml_t gene for resistance to races P and P-1. Secondly, if any investigator had chosen to increase his seed supply of Norka by harvesting only a few plants without testing the original lot, he may accidentally have developed a pure stock genetically identical with either Norka-1 or Norka-2, and thus would have observed only a uniform reaction. Thirdly, Norka wheat may have another gene for resistance apart from the Ml_t gene which is presumed to govern the reaction to all of the races studied by others. If the reaction of Norka wheat to all races except the Norka race is governed only by Ml_t, and that to the Norka race is governed by another gene,

Ml_x, the genotype is $Ml_tMl_tMl_xMl_x$ for Norka-1, and $Ml_tMl_tml_xml_x$ for Norka-2.

Alternatively, the writer may have received a stock different from that used by others. If this were the case, the need to pure-line the differential varieties and to have all workers use the same seed stocks is clear.

To determine whether the writer had the same stock or a different one, the original seed lots used by others, if available, could be tested for reaction to the Norka race. If all lots gave a non-uniform reaction then the writer must have had the same stock. Another approach would be for future workers to test both Norka-1 and Norka-2 to all races. If both Norka-1 and Norka-2 gave the same reaction, then the writer must also have had the same stock. On the other hand, if Norka-1 and Norka-2 react as they have to the Norka race, then the writer must have had a different stock.

The existence of two genetically different lines within Norka wheat is somewhat disturbing because Norka originated from a pure-line selection (presumably a single plant) of common wheat in 1908 at Akron, Colorado (1). The occurrence of two genotypic types within Norka could be explained, however, by postulating that the original plant was heterozygous for the Ml_t-ml_t or Ml_x-ml_x gene producing the difference in mildew reaction manifested by Norka-1 and Norka-2.

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CONTACT FUNGICIDES FOR PEACH SCAB CONTROL[1]

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Summary

One year of field testing indicates the possibility of controlling peach scab (Cladosporium carpophilum Thuem.) with one dormant application of Puratized Apple Spray (phenyl mercury monoethanol ammonium acetate) at the rate of 2 1/2 pints per 100 gallons of water.

INTRODUCTION

Scab is one of the most costly diseases of peaches in the southeastern States. It is an ever-present threat, and numerous applications of fungicides are necessary each season to assure control.

The conditions of fungus habit and yearly perpetuation suggest the possibility of vulnerability to eradicant chemicals.

Fruit infections begin about 2 or 3 weeks after petals fall, and may continue throughout the season. The major source of inoculum for fruit infection is from twig lesions of prior years. Twig infections probably begin in early spring. Conidia may be produced sparsely on the young twig lesions from the time of their appearance until fall, but abundant production of conidia does not occur until the following spring and early summer. During the third season, conidia are produced very sparsely, if at all, and the lesions eventually are sloughed off as the bark develops. Spore dissemination is by water under the influence of wind and gravity. Twig lesions involve only superficial areas; the fungus never penetrates to the cambium. The fungus is limited by a protective layer of cork cells that forms to separate the area of infection from adjacent normal tissue. Neither infected leaves nor fruits that overwinter on the ground are important sources of inoculum (6, 7, 8).

Dormant sprays for the control of pecan scab, Cladosporium effusum (Wint.) Demaree, have been under consideration recently (1, 2, 3, 4, 5). Only limited success has resulted on pecans, owing primarily to the rapidity of secondary spread of the disease, the fact that infected pecan leaves play an important role in overwintering of the fungus, and the difficulty in obtaining adequate coverage because of tree size. Laboratory tests with several contact fungicides have indicated fungus eradication from twigs.

This paper reports promising results in the control of peach scab by dormant sprays as a part of continuing studies on contact fungicides.

MATERIALS AND METHODS

Uniform 4-year-old peach trees of the Elberta and Redhaven varieties located at the Pontotoc Branch Experiment Station in north-central Mississippi were used for the 1959 field tests. Sprays were applied with a high-pressure hydraulic sprayer. A randomized block experimental design was employed using three-tree plots and six replicates of each variety. Care was exercised to wet the trees thoroughly when applying the dormant spray.

Both Redhaven and Elberta varieties were sprayed on the same dates. The spray dates were as follows:

Application	Date
Dormant spray	March 2, 3
1. Petal fall	March 30, 31
2.	April 13, 14
3.	April 28, 29
4.	May 14, 15
5.	May 28, 29
6.	June 12, 13

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Redhavens harvested	June 22-July 1
7. Elberta variety only	June 27
8.	July 10
9.	July 20
Elbertas harvested	July 27-August 1

The spray treatments were: 1) Puratized Apple Spray (2 1/2 pints per 100 gallons water) dormant application + captan (2 pounds per 100 gallons, 50%, wettable) on all but applications 1 and 2; 2) captan on all applications but 1 and 2 (no dormant spray); 3) Puratized Apple Spray dormant application + captan on all regular season applications; 4) captan on all regular season applications (no dormant spray); 5) wettable sulfur (6 pounds per 100 gallons) on all regular season applications (no dormant spray); 6) unsprayed check. Parathion was used as the insecticide in all treatments and was applied in all regular season applications except the one before harvest in each case.

In order to evaluate properly the dormant eradicant for scab control, the usual fungicide applications were omitted in the early portion of the season in treatments 1 and 2 to allow a period of time for scab infection. Captan was applied in the latter part of the season, however, to prevent a build-up of brown rot, since brown rot could completely destroy the fruit crop and thus ruin the opportunity of taking scab notes. Captan would not be expected to influence development of the already established scab lesions.

The unsprayed check scab ratings were not included in the statistical considerations since many of the replicates were completely destroyed by brown rot and no scab ratings could be obtained. They are included in the table of results, however, to indicate the potential high scab incidence.

Scab notes were taken by grading peaches into four arbitrary categories as to scab severity, counting the number in each category and after appropriately weighting each group, calculating scab incidence as a percentage of the weighted total. Notes were also taken on brown rot incidence as well as fruit finish, total yield, fruit size, spray injury, and so forth.

RESULTS

The results of the experiment in regard to scab incidence are given in Table 1. Where the

Table 1. Treatment means representing peach scab incidence over six replicates of a randomized complete block experimental design (three-tree plots).

Treatment	Scab incidence	
	Elberta Var.	Redhaven Var.
1. Puratized Apple Spray dormant application + captan on all but applications 1 and 2	0.07	0.03
2. Captan on all but applications 1 and 2 (no dormant spray)	1.48	4.15
3. Puratized Apple Spray dormant application + captan on all regular season applications	0.03	0.00
4. Captan on all regular season applications (no dormant spray)	0.48	0.10
5. Wettable sulfur on all regular season applications (no dormant spray)	0.38	0.93
6. Unsprayed check[a]	57.30	34.38
L.S.D. .05	1.25	0.66
L.S.D. .01	1.69	0.88

[a] Unsprayed check was not included in analysis because of high variability.

dormant spray was applied and the early season fungicide applications were omitted (treatment 1), a significant control of scab was obtained over treatment 2 where no dormant spray was applied, and the fungicide likewise omitted in early season applications. The exceptional extent of scab control achieved in treatment 1 indicates eradication of the inoculum source. The eradication appeared even more complete in this treatment in field observations since the small amount of scab that was found (as indicated in the results) came from particular sections of a few trees, indicating possible error in coverage with the eradicant chemical. The difference

of scab incidence between varieties in treatment 2 reflects a difference between the two varieties as to time scab infection begins, since both varieties were sprayed on the same dates. There was no significant influence on brown rot control by the dormant spray. Likewise, there was no evident effect on fruit finish, total yield, or fruit size. There was no evidence of toxicity. Peach leaf curl and bacterial spot were not of enough consequence in the orchard to provide reliable notes.

DISCUSSION

Further field testing will be necessary before definite conclusions can be drawn. However, the evidence indicates the possibility of eradicating the source of peach scab inoculum on peach twigs and the elimination of several regular season fungicide applications without the danger of scab increase. The results in this experiment could have been influenced somewhat by residual action of the dormant fungicide as well as by the eradicant action. However, this is not believed to be true in view of previous laboratory studies (5) and the 2-month period of weathering between the dormant application and the first captan application (treatment 1) -- a period during which 8.88 inches of rainfall was recorded at the local station. The influence of the dormant spray on other diseases of importance on peaches, such as leaf curl and bacterial spot, is yet to be determined. If the results reported here are confirmed in subsequent field trials, the way will be opened to many new approaches for study and the possible use of many fungicides that would otherwise be limited by the Food and Drug Administration restrictions. Several compounds show promise for use in this respect and are under consideration, especially those belonging to the quaternary nitrogen group that exhibit detergent properties as well as fungicidal activity.

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HYPOXYLON CANKER ON EUROPEAN ASPEN

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Abstract

Cankers caused by Hypoxylon pruinatum were found on Populus tremula in Wisconsin. The susceptibility of European aspen indicates that it cannot be considered a source of resistance in crosses with native species. No previous reports of infection on European aspen in North America or western Europe were found. It has been reported in Russia.

Hypoxylon canker, caused by Hypoxylon pruinatum (Klotzsch) Cke., is the most serious disease of quaking aspen (Populus tremuloides) and bigtooth aspen (P. grandidentata). Development of resistance to this disease is an important objective of any tree genetics program that involves use of these native aspens as parent materials. European aspen (P. tremula) is sometimes used as a parent in crosses with native aspens. For this reason, evaluation of the species as a possible source of resistance to the disease is important. During the past 3 years several Hypoxylon cankers have been found on the progeny of selections of European aspen under field test in the tree improvement program of The Institute of Paper Chemistry in Wisconsin. Symptom expression on European aspen is identical with that of cankers on quaking aspen (1). The conidial and perfect stages of the fungus also are identical on the P. tremula and the P. tremuloides cankers. A comparison of cultures isolated from the two host species further confirmed that H. pruinatum was causing the cankers on the European aspen.

The prevalence of infection on European aspen appears to be comparable to that on quaking aspen of the same age. P. tremula as a parent in crosses with native aspens cannot be considered as a source of Hypoxylon canker resistance unless specific selections are found that have demonstrated resistance to this disease.

A search of the literature shows that P. tremula has never been reported as a host of H. pruinatum in North America, nor does it appear to have ever been reported as a host in any western European countries. The disease appears to be present, however, on P. tremula in the Soviet Union. Ankudinov (2), in a paper on decay in aspen, reviews a paper by Semjakin (3) reporting the results of a study of black canker of aspen caused by H. holwayi Ell., which is a synonym for H. pruinatum. The prevalence of infection mentioned in this review would seem to indicate that the disease is at least as prevalent as it is on P. tremuloides in North America. Unfortunately, the authors have not had access to Semjakin's (3) paper. Although this paper was written in 1936, no later Russian papers on Hypoxylon canker have been found.

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FUNGI ASSOCIATED WITH DEFOLIATION OF TAXUS CUTTINGS[1]

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Abstract

As part of a project on defoliation of Taxus cuspidata nana, cuttings were rooted in a propagating bed in a greenhouse. High mortality of the cuttings was found to be associated with a Fusarium species and Rhizoctonia solani.

As part of a project on defoliation of Taxus cuspidata nana, 100 long-shoot cuttings were cut at random from field stock in a nursery in Newport, Rhode Island, December 16, 1958. Almost all the plants were shedding to some degree; possibly 1 to 2 percent of the needles had dropped. The leaves which were being shed had turned yellow from the base upward.

The weather was unseasonably cold during November and December 1958, but the plants showed no evidence of cold injury or desiccation.

The cuttings were on the average about 10 inches long. The leaves were removed from the lower 4 inches of each cutting. A small piece was cut from the basal end and a small piece of bark was removed from opposite sides to expose fresh cambial surfaces. The cut ends were then dipped into a rooting hormone powder.

Half the cuttings were immersed in a suspension of captan (N-trichloromethyl thiotetrahydrophthalimide) 50W containing 22 grams per gallon, and half were left untreated. At 10-day intervals the treated cuttings were drenched with captan at the same concentration, using 1 quart of drench per square foot of bed.

The cuttings were stuck into a propagating bed filled with sand and shaded with dark green gauze. Forty inches of sand separated the two lots of cuttings. Before it was used, the bed containing the sand was sterilized with a steam coil for 2 hours. Bottom heat in the bed was maintained at 64° F. For the period December 16-March 1 the day temperatures in the greenhouse averaged 76° for the maximum, and 60° for the minimum. Night temperatures averaged 74° maximum and 62° minimum.

By January 12 the untreated cuttings were shedding more than the treated ones, but 10 days later there was very little difference between the two lots. At this time most of the buds had broken dormancy and were growing, and callus tissue had formed at the base of the cuttings.

By February 26 many of the cuttings had died. The mature leaves had turned a bright brown and shed, and the new growth had wilted and died. The shedding leaves did not show the characteristic yellowing from the base upward, which has been observed so often under field conditions. Eighteen plants in the treated lot remained alive and 13 of the controls. These appeared to be healthy, with normal root systems. They were transferred to pots on March 2 and have continued to grow well.

Since the symptoms suggested something more than a physiological disorder, diseased tissues were plated on potato-dextrose agar, using aseptic techniques. A Fusarium sp. and Rhizoctonia solani were isolated from wood of affected plants and identified.

Further work needs to be done to determine the prevalence, pathogenicity, and control of these fungi on Taxus in propagating houses and whether they are limited to T. cuspidata nana or may also attack other varieties.

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A NEW INSECT VECTOR OF SQUASH MOSAIC VIRUS[1]

W. R. Sitterly[2]

Abstract

The banded cucumber beetle (Diabrotica balteata) was demonstrated to be a new and efficient vector of squash mosaic virus in the commercial truck crop area of Coastal South Carolina.

Along with the spotted and striped cucumber beetles, the banded cucumber beetle transmitted the virus after only 5 minutes' feeding on infected tissue, and continued transmission for at least 4 consecutive days without returning to feed on infected leaves. No prolonged incubation period in the body of the beetle was required.

INTRODUCTION

Within the past 10 years squash mosaic has become progressively more prevalent in the commercial truck crop areas of Coastal South Carolina. Although a successful spring crop of summer squash is possible, the summer vector build-up makes it very difficult to grow a fall crop. As part of a study on the nature and control of this disease, an investigation was initiated to determine the specific insects responsible for the transmission of the virus, the length of the incubation period (if any) within the insect, and the length of time that a specific insect may continue to transmit the virus after it has fed on a diseased plant.

MATERIALS AND METHODS

Green peach aphids (Myzus persicae) and three species of cucumber beetles common in Coastal South Carolina were captured in the field. These species were the spotted cucumber beetle (Diabrotica undecimpunctata howardii), the banded cucumber beetle (Diabrotica balteata), and the striped cucumber beetle (Acalymma vittata). After capture, the insects, except for those used in treatment No. 1, were held for 10 days on non-susceptible plants, and then starved for 24 hours before they were placed on diseased squash plants. Previous tests had indicated that 10 days were adequate to insure that all insects were virus-free.

Transmission tests were conducted in 18 x 18 x 36-inch insect cages into which potted plants of the Yellow Crookneck summer squash variety were placed. The plants were allowed to grow to a stage of lateral cotyledonary development before being subjected to insect feeding. The insects, in colonies of five, were allowed a 24-hour feeding period on test plants before being removed. Host plants were then allowed to develop for 30 days. Work was done in a screened greenhouse where the temperature varied from 70° to 85° F.

Treatments were as follows: 1) insects taken directly from field to plant; 2) insects allowed to feed 5 minutes on diseased leaves, then placed on healthy plants; 3) insects allowed to feed 3 hours on diseased leaves, then placed on healthy plants; 4) insects allowed to feed 24 hours on diseased leaves, then placed on healthy plants; 5) insects allowed to feed 3 hours on diseased leaves, starved 24 hours, then placed on healthy plants; 6) insects allowed to feed for 24 hours on diseased leaves, starved 24 hours, then placed on healthy plants; 7) insects allowed to feed 24 hours on diseased leaves, then transferred daily to healthy plants for 6 consecutive days. All treatments were replicated six times.

RESULTS AND DISCUSSION

Cucumber beetles of all three species tested were found to be viruliferous at the time of their capture in the field. After the beetles were freed of the virus by holding them for 10 days on non-susceptible plants, all three species were again able to transmit the virus to susceptible plants after only 5 minutes' feeding on infected plants (Table 1). Longer periods of feeding, however, appeared to result in more efficient virus transmission, since feeding times of 5

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minutes, 3 hours, and 24 hours resulted in 47, 55, and 70 percent infection, respectively, on inoculated squash plants. The virus was acquired during a 24-hour feeding period and was transmitted to healthy squash plants during the succeeding 24-hour period. Thus the virus incubation or latent period within the body of the beetle must be less than 48 hours. All three species of beetles continued to transmit SMV for at least 4 days without returning to feed on infected plants, but none transmitted the virus longer than 5 days. The green peach aphid (Myzus persicae) did not transmit the virus to healthy plants, even after the aphids had fed on diseased plants.

Table 1. Transmission of squash mosaic virus by banded, spotted, and striped cucumber beetles, and by the green peach aphid, expressed as the percentage of exposed plants which later showed symptoms of mosaic.

Treatment	Percentage of plants showing symptoms of mosaic transmitted by:			
	Spotted cucumber beetle	Banded cucumber beetle	Striped cucumber beetle	Green peach aphid
Direct from field to plant	50	35	50	0
Feed 5 min., put directly on host	45	50	45	0
Feed 3 hrs., put directly on host	55	60	55	0
Feed 24 hours, put directly on host	70	80	60	0
Feed 3 hours, starve 24 hours, put on host	50	90	35	0
Feed 24 hours, starve 24 hours, put on host	70	60	50	0
Six-day transmission:				
1st. day	35	100	90	0
2nd. day	35	90	90	
3rd. day	90	45	35	
4th. day	45	35	45	
5th. day	35	0	0	
6th day	0	0	0	

This is the first instance in which SMV has been shown to be transmitted by the banded cucumber beetle. This beetle occurs mainly in the southeastern States, particularly at the lower elevations, and has been reported by entomologists to be increasing in numbers. Currently it is the most prevalent of the three species in Coastal South Carolina.

Retention of the virus by the vector for at least 4 days after feeding on a susceptible plant is undoubtedly one factor which permits the rapid spread of the virus under field conditions. These results are in partial agreement with those of Freitag[3] who reported that a small percentage of individual beetles retained the ability to transmit the virus for as long as 20 days when permitted a 15-day acquisition-feeding period. Perhaps utilization of the longer feeding periods used by Freitag would have resulted in longer transmission periods for individual beetles. The lack of an extended incubation or latent period within the body of the vector confirmed the work of Freitag, who indicated that this latent period must be less than 10 hours.

SOUTH CAROLINA AGRICULTURAL EXPERIMENT STATION, CLEMSON

[3]Freitag, J. H. 1956. Beetle transmission, host range, and properties of squash mosaic virus. Phytopathology 46:73-81.

DODINE-CAPTAN COMBINATION CONTROLS BACTERIAL SPOT OF PEACH[1]

Urban L. Diener and C. C. Carlton[2]

Abstract

Sprays of dodine, sulfur, and captan used in varying combinations with insecticides were applied to Elberta peach trees in Alabama in 1959 for the control of bacterial spot. Dodine-captan combinations reduced bacterial spot significantly compared with sulfur or captan alone. No symptoms of phytotoxicity were noted after use of dodine with captan. Captan also appeared to have a synergistic effect when used with dodine.

INTRODUCTION

Bacterial spot (bacteriosis), caused by Xanthomonas pruni (E. F. Sm.) Dows., has been the most destructive disease of peach in Alabama in recent years because there has been no effective control. Experiments to control this disease by chemical and antibiotic sprays have been conducted in Alabama since 1953.

The first indication of experimental control resulted from applications of dodine and sulfur to Elberta peach trees in 1958[3]. Dodine is the coined common chemical name of n-dodecylguanidine acetate, manufactured as Cyprex dodine 65-W by the American Cyanamid Company. In this experiment a reduction in diseased fruit from 20.3 percent in plots sprayed with captan and sulfur to 10.5 percent in plots sprayed with dodine-sulfur combination indicated that dodine reduced bacterial spot. The investigation was continued in 1959, using different concentrations of dodine combined with sulfur and/or captan in search of a more effective spray combination.

MATERIALS AND METHODS

Experimental plots in the Elberta peach orchard at the Chilton Area Horticultural Substation, Clanton, Alabama consisted of five to seven trees, replicated four times in a randomized block design. The following combinations of fungicides and insecticides with rates on a 100-gallon basis were evaluated in 1959.

1. Dodine (65%) 1 1/2 lb. + malathion (25%) 3 lb.
2. Dodine (65%) 1 lb. + sulfur (80-90%) 6 lb. + malathion (25%) 3 lb.
3. Dodine (65%) 1 1/2 lb. + sulfur (80-90%) 3 lb. + malathion (25%) 3 lb.
4. Dodine (65%) 1 lb. + captan (50%) 1 lb. + parathion (15%) 2 lb.
5. Dodine (65%) 1 1/2 lb. + captan (50%) 1 lb. + sulfur (80-90%) 3 lb. + parathion (15%) 2 lb.
6. Dodine (65%) 1 lb. + captan (50%) 1 lb. + sulfur (80-90%) 3 lb. + Sevin (50%) 2 lb.
7. Captan (50%) 2 lb. + parathion (15%) 2 lb.
8. Sulfur (80-90%) 6 lb. + parathion (15%) 2 lb.

Dormant spray of concentrated lime-sulfur (1:16) for peach leaf curl and scale was applied January 16, 1959. The remaining sprays were applied on the following dates: bloom, March 30; covers, April 9, 17, and May 1; preharvest, May 15, 27, June 3, 15, and 30. Data on phytotoxicity and bacterial leaf spot were taken on June 25 and July 21. The number of diseased fruit were counted for all peaches harvested on July 17 or a minimum of 200-250 per tree on each of five trees in a plot. Fruit with one or more lesions or spots were counted as diseased. Approximately 1000 peaches were counted for each plot. Fruit counts were summarized in percent of clean and diseased fruit and the replications averaged.

Single tree applications of treatments No. 2 (dodine-sulfur-malathion), and No. 8 (sulfur-parathion) were made to trees in three different varietal blocks of two trees each. These three breeding lines of early peaches were moderately to highly susceptible to bacterial spot. The last sprays were applied June 3 and the data summarized from all fruit counted on June 17, 19, 22, and 24.

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[1]Published with the approval of the Director of the Alabama Agricultural Experiment Station.

[2]Respectively, Associate Plant Pathologist and Superintendent, Chilton Area Horticultural Substation, Auburn University Agricultural Experiment Station.

[3]Diener, Urban L. 1959. Fungicidal control of bacterial spot of peach in Alabama in 1958. J. Alabama Acad. of Sci. 31: 2.

RESULTS AND DISCUSSION

Phytotoxic effects from the dodine 1 lb.-sulfur 6 lb. treatment were not observed in 1958. However, dodine and dodine-sulfur combinations resulted in severe foliage injury in 1959 (Table 1). This was observed primarily as severe shot-holing of the leaves and slight defoliation. Except on treatment Nos. 1, 2, and 3, there was no shot-holing or other evidence of phytotoxicity from any treatment. The occurrence of typical bacterial spot lesions and shot-holing is footnoted on Table 1. Severe yellowing and defoliation from red spider injury in Sevin-sprayed plots (treatment No. 6) was observed on July 21.

Table 1. Phytotoxicity to foliage (index rating) and control of diseases of peach (percent of fruit diseased) by spray applications with several combinations of fungicides in Alabama in 1959.

Treatment (lb./100 gal)	Foliage Index[a]	Mean Percent (4 Reps.) Bact. Spot		Peach Scab	Brown Rot
1. Dodine 1½ + Malathion 3	3.0	3.0		+[b]	0.1
2. Dodine 1 + Sulfur 6 + Malathion 3	1.5	2.9	*	0	0.3
3. Dodine 1½ + Sulfur 3 + Malathion 3	2.5	3.8		0	0.3
4. Dodine 1 + Captan 1 + Parathion 2	0.0	1.0		0	0.1
5. Dodine 1½ + Captan 1 + Sulfur 3 + Parathion 2	0.0	1.0	**	0	+
6. Dodine 1 + Captan 1 + Sulfur 3 + Seven 2	0.3[c]	3.1		0.1	0.1
7. Captan 2 + Parathion 2	0.5[c]	4.9		+	0.1
8. Sulfur 6 + Parathion 2	0.5[c]	6.5		+	0.1

Statistically significant reduction at (.05)*, (.01)** [d]

[a] Leaf damage rating: 0 -- none, 1 -- slight, 2 -- moderate, 3 -- severe.

[b] + represents less than 0.05 percent.

[c] Rating due to slight foliage injury from bacterial spot in one or more applications.

[d] Group comparisons of Nos. 1, 2, and 3 versus Nos. 7 and 8; Nos. 4, 5, and 6 versus Nos. 7 and 8.

The addition of 1 pound of captan apparently had a safening effect on dodine, as no shot-holing was noted on the foliage of treatment Nos. 4, 5, and 6. In treatment No. 5, the level of dodine was 1 1/2 pounds but still no phytotoxic effects were observed.

Reduction in the percent of fruit infected with bacterial spot in dodine plots can be noted in Table 1. Incidence of bacterial spot in 1959 was considerably lower than in 1958 in the same orchard (approximately 6 percent as compared with 20 percent). Overall differences in the reduction in disease incidence were not statistically significant by the F test. However, when group comparisons were made, significant results were obtained although the contrasts were not independent. Treatments containing dodine or dodine-sulfur (Nos. 1, 2, and 3) as a group were statistically significant at the 5 percent level when compared with the group of the two check treatments (Nos. 7 and 8). However, treatments containing dodine-captan or dodine-captan-sulfur (Nos. 4, 5, and 6) were statistically significant at the 1 percent level in group comparisons with the check treatments (Nos. 7 and 8) containing only captan or sulfur. Thus, it would appear that the addition of captan resulted in not only a safening action in reducing the phytotoxic effects of dodine to the foliage, but it also appeared to have a synergistic effect whereby an increase in disease control was obtained. It was assumed that malathion and parathion had no bactericidal or phytotoxic effects in the treatment combinations.

Control of peach scab and brown rot by all treatments and treatment combinations was excellent. Six unsprayed trees in an adjoining row showed 18.7 percent of fruit with bacterial

spot, 24.2 percent with peach scab, and 4.8 percent with brown rot.

Approximate t-tests were applied to data from dodine-sulfur-malathion versus sulfur-parathion treatments on single tree plots. The effectiveness of dodine in reducing bacteriosis on these more susceptible varieties was significant at the 1 percent level of confidence in each case. A summary of the data from the three breeding lines specified B, C, and D showing the percent of fruit with bacterial spot for each treatment is as follows:

Treatment	B	C	D
Sulfur-parathion	63.1	11.1	17.1
Dodine-sulfur-malathion	17.4**	2.3**	11.1**

**Significant at (.01) level by approximate t-test.

With all three varieties, the dodine 1 pound-sulfur 6 pound treatment reduced the number of bacterial spot-infected fruit substantially.

CONCLUSIONS

Based on 2 years' data, it was concluded that dodine at a 1-pound rate combined with sulfur reduced bacterial spot of peach, but with phytotoxic effects to the leaves as evidenced by shot-holing. One year's data on combinations of dodine and captan showed that captan eliminated the phytotoxic effects of dodine. It appeared that captan also increased the effectiveness of dodine in bacterial spot control. Thus, captan displayed a synergistic as well as a safening effect, when used in combination with dodine as a spray for the control of bacterial spot of peach.

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DEATH OF DORMANT BUDS IN SWEET CHERRY[1]

H. Ronald Cameron[2]

Summary

A death of dormant buds in sweet cherry is ascribed to a strain of Pseudomonas syringae. Symptoms on sweet cherry appear to be limited to a killing of dormant buds and do not seem to be associated with the formation of leaf spots or cankers. Of twelve chemicals applied, Bordeaux and Puratized Agricultural Spray were the most effective for control.

INTRODUCTION

Since 1953 the condition known as "dead-bud" has been a serious disease of sweet cherries in western Oregon. The killing of buds was described by F. L. Griffin in 1911 (5) and was subsequently investigated by H. P. Barss (1, 2, 3). Barss and numerous other workers reporting on bacterial canker of stone fruits have frequently mentioned the killing of buds as one of the symptoms (4, 8, 9), but the killing of buds has been considered of minor importance and seldom, if ever, of economic significance. Under the climatic conditions of the Willamette Valley, however, the death of dormant buds became so serious that growers were forced to remove some affected orchards and the cherry-brining industry considered the disease a threat to continued operation. Loss of 70 percent of the flower and leaf buds during a year was not uncommon and, after several such years, retention of the affected orchard was not economical.

SYMPTOMS

"Dead-bud" is primarily a disease of mature trees, but symptoms have been found on occasional 3-year-old trees. Symptoms were usually noticed first in the lower part of the tree and in succeeding years were found higher in the tree also. This sweet cherry disease usually occurred first in an orchard either adjacent to a diseased cherry or pear orchard, or it appeared in areas of an orchard where there was poor air drainage. Incidence of infection of trees isolated from other trees, as in the outside row of an orchard, frequently remained low as contrasted with high disease incidence in the center of the orchard. The trees, Figures 1A and 1B, were photographed on the same day in healthy and diseased areas of the same orchard.

The first specific symptom is a brown discoloration within buds during late February and mid March. The disease then appeared as a dark-brown area around the base of the bud scales and eventually spread through the entire bud. Occasionally the discoloration extended into the cortex of the supporting spur or terminal shoot, but cankers were not formed. If all of the buds on a terminal or spur were killed in one season that terminal or spur died the following growing season. Dead buds usually remained on the tree indefinitely but could be easily brushed off. Usually not all of the buds on a given terminal were killed. The one or two remaining healthy buds produce long vigorous shoots due to the reduced number of growing points. Therefore, successive seasons of severe infection result in long, almost bare branches with scattered vigorous terminal shoots.

Failure of buds to open gave diseased trees their most characteristic appearance in the spring (Fig. 2). Affected trees normally retained their vigor for from 5 to 6 years but slowly declined because of reduced leaf area. Yield was often reduced by one-half within the first 3 years and pickers refused to pick the remaining scattered crop.

ETIOLOGY

The symptoms were originally attributed by the growers to various climatic and soil conditions. Investigations during 1955 and 1956 indicated that the accumulation of lead arsenate in the soil was not a possible cause of "dead-bud." Neither boron (applied as a fall and spring spray of Poly-bor) nor zinc (applied as zinc sulfate in 1956 and zinc chelate in 1957-58) applied over a 3-year period gave any control. Various combinations of nitrogen, potassium sulfate, treble

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[1]Technical Paper No. 1286, Oregon Agricultural Experiment Station.

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FIGURE 1. A -- Severe "dead-bud" on sweet cherry tree in the center of the orchard. Note lack of leaves and flowers in the lower two-thirds of the tree. B -- Comparable healthy tree.

FIGURE 2. The effect of "dead-bud" on the previous season's terminal growth. All the buds not open were dead.

super phosphate, and lime were applied in cooperation with Dr. O. C. Compton of the Horticulture Department. None of the fertilizer applications had any effect on the disease incidence. Thus, neither soil deficiencies nor low fertility seem to have any association with the "dead-bud" disease. Weather data from 1954 through 1959 (6, 7) were reviewed for western Oregon and gave no reason to suspect late frosts or other climatic conditions as the primary incitant. However, disease incidence and severity seemed to be directly correlated with seasonal variations of weather. The average monthly temperature for the months from November through April appeared to be of prime importance in determining the severity of symptoms the following spring. The longer that temperatures were between 35° and 43° F the more severe were the symptoms. No definite correlation could be detected between either the amount of rainfall, number of days of rainfall, number of days below 32° F, monthly maximum temperatures or monthly minimum temperatures. The lack of correlation with amount of rainfall may be explained by the more than ample rainfall during this period in all of the years recorded.

Pathogenic fungi have not been isolated consistently from diseased buds. Young cherry trees inoculated with budwood from diseased trees gave no indication of virus transmission of "dead-bud" after 4 years. There was no indication that the "dead-bud" symptoms were caused by either a virus or a fungus.

BACTERIA

During the spring of 1956 isolations frequently yielded a white bacterium. Subsequent isolations and inoculations in 1957 and 1958 proved this bacterium was responsible for "dead-bud." Host range tests and culture experiments indicated that the bacterium was a species of Pseudomonas and would fall within the limits of variation generally accredited to Pseudomonas syringae. Minor differences from P. syringae were noted in the host range and in the cultural characters of the bacterium. The "dead-bud" bacterium was not pathogenic on lemons, caused only mild necrosis on lilac, but incited typical P. syringae symptoms on cowpea and broad bean stems, clover foliage, and on pear fruit. All isolates would kill buds on Prunus species but only very rarely would an isolate also incite cankers. The form, texture, and coloration of colonies in solid media (Starr's medium "B")[3] closely resembled P. syringae. The "dead-bud" bacterium was capable of growing in a 7 percent sodium chloride solution while P. syringae normally would not grow in a more than 6 percent salt solution. Since P. syringae has already been reported as a pathogen causing leaf spots, cankers and a killing of buds on cherry it will be convenient from the pathologist's viewpoint to speak of the new isolate as the "dead-bud" strain.

CONTROL

Exploratory experiments in 1955-56 suggested the feasibility of controlling the disease by sprays. During the 1956-57 and 1957-58 seasons extensive experiments were set up in two sweet cherry orchards. Individual plots consisted of four trees with one of the four selected at random as an unsprayed check. Six replications were run in each of the two orchards. All plots were sprayed with an hydraulic sprayer operated at 500 pounds' pressure and a number 7 disc was used in the spray gun. Large trees received 15 to 20 gallons of spray and the average trees received 10 to 15 gallons. Twenty milliliters of spreader-sticker per 100 gallons of spray were used with all materials except Bordeaux. Results were recorded as the number of dead buds out of a 150-bud sample selected at random from 1-year-old terminals. Tables 1 through 4 give the treatments, dates of application, and results for the experiment in both orchards for the 1956-57 and 1957-58 seasons.

Since most of the unsprayed check trees were surrounded by sprayed trees an effort was made to determine if they were affected by the close proximity of the sprayed trees. This was determined by taking readings at increasing distances from the sprayed area. Three hundred buds were counted on each tree (Table 5).

From Table 5 it is apparent that the check trees in the sprayed part of the orchard had a lower number of dead buds than the trees outside the experiment. Since the check trees were randomly placed on all sides of the sprayed trees, it was not possible to account for the lower infection by drift of spray materials. Rather it was thought that the amount of inoculum had been reduced in the sprayed area and therefore all of the checks were not as heavily infected as the trees outside the sprayed area. Because of this discrepancy between the checks in the plots

[3]Media formula obtained from correspondence with Dr. Harley English.

Table 1. Effectiveness of different spray applications for control of dead-bud in orchard number 1, 1956-57 season.

Treatment	Mean[a]	5%[b]	1%	p[c]
Streptomycin 200 ppm, plus bordeaux 8-8-100 on July 17, 1956, and streptomycin plus 16-16-100 bordeaux on October 3, 1956, and February 20, 1957.	8.06			
Bordeaux 8-8-100 on July 17, 1956, followed by 16-16-100 on October 3, 1956, and February 20, 1957.	11.61			2
Puratized Agricultural Spray 1 pint per 100 gallons on July 17, 1956, October 4, 1956, and February 19, 1957.	31.67			3
Copper A 1-1/2 lbs per 100 gallons on July 16, 1956, October 4, 1956, and February 20, 1957.	46.61			4
Streptomycin 200 ppm on July 16, 1956, October 2, 1956, and February 18, 1957.	76.72			5
Unsprayed check	79.78			6
Streptomycin 100 ppm on October 2, 1956, October 14, 1956, and February 18, 1957.	82.83			7

[a]Mean is mean number of dead buds in 150 bud samples per tree adjusted to the "Y" means, all replications included.
[b]Any two means not bracketed by the same line are significantly different at the respective percentage level.
[c]Position or rank of treatment.

Table 2. Effectiveness of different spray applications for control of dead-bud in orchard number 2, 1956-57 season.

Treatment	Mean[a]	5%[b]	1%	p[c]
Bordeaux 8-8-100 on August 8, 1956 followed by 16-16-100 on October 10, 1956.	1.89			
Streptomycin 200 ppm, plus bordeaux 8-8-100 on August 8, 1956 and 16-16-100 on October 8, 1956.	2.61			2
End-o-thol - 2 quarts per 100 gallons applied August 7, 1956, followed by bordeaux August 17, 1956 and October 8, 1956	4.39			3
Puratized Agricultural Spray 1 pint per 100 gallons on August 8, 1956 and October 5, 1956.	4.94			4
Iodophor - 1 pt. per 100 gallons on October 11, 1956.	6.00			5
Copper A 1-1/2 lbs per 100 gallons on August 8, 1956 and October 5, 1956.	35.89			6
Merthiolate (Eli Lilly) - 1/3 lb per 100 gallons on October 11, 1956.	44.78			7
Experimental Quaternary 3104 (Rohm & Haas) on August 8, 1956 and October 11, 1956.	46.67			8
Check	52.79			9
Streptomycin 100 ppm on August 8, 1956 and October 8, 1956.	57.17			10

See Table 1 for footnotes.

Table 3. Effectiveness of different spray applications for control of dead-bud in orchard number 1, 1957-58 season.

Treatment	Mean[a]	5%[b]	1%	p[c]
Puratized Agricultural Spray 1 pint per 100 gallons on July 23, October 9, 1957 and February 27, 1958.	7.36			
Bordeaux 12-12-100 on July 24, 1957 and October 11, 1957.	12.82			2
Bordeaux 8-8-100 on July 25, 1957 followed by 16-16-100 on October 10, 1957 and 8-8-100 on February 27, 1958.	16.96			3
Bordeaux at 8-8-100 on July 25, 1957 and October 11, 1957 applied 4-4-100 on March 1, 1958.	26.91			4
Agrimycin 500 (42% copper) 5 lbs per 100 gallons applied July 23, 1957, October 9, 1957 and February 27, 1958.	33.42			5
Bordeaux 12-12-100 on July 24, 1958.	34.73			6
Check	106.20			7

See Table 1 for footnotes.

Table 4. Effectiveness of different spray applications for control of dead-bud in orchard number 2, 1957-58 season.

Treatment	Mean[a]	5%[b]	1%	p[c]
Puratized Agricultural Spray 1 pint per 100 gallons on August 21, 1957 and October 30, 1957.	5.96			
Omadine (Olin Mathieson 42 percent copper salt) 1 lb per 100 gallons on August 21, 1957 and October 31, 1957.	6.89			2
Merthiolate 1/3 lb per 100 gallons on August 21, 1956 and October 30, 1957.	8.00			3
C.O.C.S. 4 lbs per 100 gallons on August 22, 1957 and November 1, 1957.	8.32			4
Bordeaux 8-8-100 on August 23, 1957 followed by 16-16-100 on October 31, 1957.	11.77			5
Bordeaux 12-12-100 on August 23, 1957 and October 31, 1957.	13.31			6
TAG 1/2 pint per 100 gallons on August 21, 1957 and October 30, 1957.	19.27			7
Bordeaux 8-8-100 on August 23, 1957 and November 1, 1957.	20.34			8
Check	44.52			9

See Table 1 for footnotes.

and the much higher disease incidence outside the sprayed area, it is evident that the control measures were in reality considerably more effective than indicated by Tables 1-4.

Table 5. The effect of distance from the sprayed area on number of buds killed.

Location of tree	Number of buds dead out of 300 sampled
Sprayed tree	4
One tree away from sprayed tree (equivalent of unsprayed checks)	45
Two trees away from sprayed tree	161
Three trees away from sprayed tree	178
Four trees away from sprayed tree	tree missing
Five trees away from sprayed tree	242

Although in the second orchard in 1957-58 the majority of treatments were not significantly different because of variation within the plots, they appeared in approximately the same order of effectiveness as in the first orchard. Results in the second orchard were considered a valid indication of the relative value of the materials tested.

The results from these tests favored some of the copper and mercury compounds. This was anticipated since they were among the more active bactericides. Ferbam and Acti-dione had been used in earlier tests and did not give any control. Although streptomycin was very effective in the laboratory, it was not satisfactory under field conditions, probably because of its comparatively poor weathering qualities. Streptomycin mixed with Bordeaux or with a fixed copper did not materially increase the effectiveness of either material. The slight improvement certainly could not justify the cost of the streptomycin.

Satisfactory control has been possible with either Bordeaux or Puratized Agricultural Spray used with a spreader-sticker. In severely affected orchards it was necessary to apply sprays for several years before yields increased. This lag in yield increase was attributed to the 2 or 3 years required to establish a new spur system in the tree. With careful application, severely diseased orchards may be returned to good commercial production in 3 to 4 years.

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NATURAL HOSTS OF CUCUMBER MOSAIC VIRUS IN ISRAEL[1]

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Summary

In the course of a survey for the identification of causal agents of mosaic diseases in Israel, cucumber mosaic virus was isolated from sugar beet, common beet, charlock, shell-flower, potato, safflower and Cavendish banana. The identity of the virus was established by its host range and by cross-protection tests against a strain of cucumber mosaic virus causing necrotic local lesions on zinnia.

INTRODUCTION

Cucumber mosaic virus (CMV) has been identified in Israel in several cultivated plants, for example, fodder beet, cucumber, melon, vegetable marrows, tomato, pepper, eggplant, and periwinkle (6). A continuation of the survey for the identification of the viruses causing mosaic diseases in Israel resulted in the discovery of some additional natural hosts of CMV.

IDENTIFICATION METHODS

Test plants were kept in a screened greenhouse and fumigated at regular intervals throughout the year. All test plants were sap-inoculated in the early stages of growth, and 400 mesh carborundum was used as an abrasive in the transfers. Banana leaves were inoculated by the quick inoculation method (7) without addition of phosphate, but results were erratic. Satisfactory inoculum was obtained by disintegrating small pieces of banana leaves in a Waring Blendor, with the addition of a little water or phosphate buffer adjusted to pH 7.

The first series of inoculated test plants were: Chenopodium amaranticolor, Cucumis sativus (cucumber) - Beit Alfa variety, Datura stramonium, Gomphrena globosa, Nicotiana glutinosa, Physalis floridana, Solanum villosum, Zinnia elegans - Will Rogers variety. Several viruses isolated from cucurbits in Israel will be discussed in another paper; of them only CMV has been found to affect N. glutinosa. Among the viruses that might have affected both cucumbers and N. glutinosa, alfalfa mosaic virus may be differentiated from CMV by the different reaction of C. amaranticolor to them (2). Tobacco ringspot virus may be distinguished from CMV by the different reactions of cucurbits, Solanaceae (5) and C. amaranticolor (1, 2). When symptoms on the test plants indicated the probable presence of CMV, the inoculum recovered from cucumber and N. glutinosa was tested on a similar series of plants. In this way the reaction of the test plants was investigated, after other viruses which might have been involved in the first series were eliminated by passage through cucumber or N. glutinosa. The identification was concluded by a cross-protection test on zinnia (Will Rogers variety), against the local lesion strain of CMV (6).

The reaction of the different test plants to inoculation with isolates from the hosts mentioned following was found to correspond with that already described for CMV isolates encountered in Israel (6). In no case was the strain causing necrotic local lesions on zinnia encountered, and a high protection was always secured against this strain by the isolates. The possible presence of other viruses is not discussed here.

HOST PLANTS OF CMV

SUGAR BEET (Beta vulgaris): Symptoms on this crop are similar to those described for fodder beet (6). Infected material was collected in one case in the western Jizreel Valley, in June 1959. The author has no explanation for the absence of this disease on beets in the remaining regions in Israel.

COMMON BEET (Beta vulgaris): This weed was often observed to be affected by a mosaic similar to that described on fodder beet (6). Plants showing these symptoms were commonly observed during the winter of 1958-1959 in the Huleh and Jordan Valleys and in the Coastal Plain.

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[1] Publication of the Agricultural Research Station, Beit Dagan - Rehovot. 1959 Series, No. 301-E.
[2] Plant Pathologist.

CHARLOCK (Sinapis arvensis): This is one of the most common winter weeds in Israel. Foliage presented a brilliant yellow mosaic, there was reduction in leaf size, generally accompanied by severe leaf malformation and puckering. During the winter of 1958-1959 plants showing these symptoms were found in the Huleh Valley and the Jordan Valley, in and near fields of cultivated crops affected by CMV.

SHELL-FLOWER (Molucella laevis): Shell-flower is a common spring and summer weed in cultivated fields in Israel. Brilliant mosaic and puckering were observed in one case in the Coastal Region in May 1959.

POTATO (Solanum tuberosum): The virus was recovered in October 1958 from 2-month-old Up-to-date potatoes in one farm in the Upper Galilee. Foliage symptoms were a brilliant yellow mottle, necrotic spotting and puckering. No tuber symptoms were observed, and disease transmission through the tubers (3) was not investigated although the virus was recovered from tubers collected from infected potatoes. Disease incidence was very high only in the part of the field near mosaic-infected cucurbits and fodder beets.

SAFFLOWER (Carthamus tinctorius): The virus produces a mild mosaic on foliage and on the involucral bracts. This disease was observed several times in May 1958 in the western Jizreel Valley, and there was little damage to the crop. Similar symptoms were observed again in the spring of 1959 in the Coastal Region, but virus identification work was not then carried out.

CAVENDISH BANANA: Mottle is very evident on the youngest leaves, as chlorotic narrow stripes (Fig. 1). In some cases these stripes turn necrotic, tissues disintegrate and the young leaves are torn along the secondary veins. The leaves are generally narrower and shorter than those of healthy plants. Symptoms tend to fade with age. Sometimes severe plant stunting was observed. Heart rot symptoms were observed, but no clear correlation between this symptom and CMV (4) was established. The disease was identified in several plants in banana plantations at Herzlia, in the Coastal Region, during the winter of 1958-1959.

FIGURE 1. Symptoms of cucumber mosaic virus on Cavendish banana.

CONCLUSION

The results of the survey as summarized here and elsewhere (6) indicate that cucumber mosaic virus has a wide host range among cultivated and wild plants in Israel, including plants belonging to the Chenopodiaceae, Cruciferae, Apocynaceae, Labiatae, Solanaceae, Compositae, and Musaceae.

This disease causes severe losses in all parts of the country in spring and autumn, but is of minor importance in summer. In winter the principal economic hosts, cucurbits, pepper and tomato, are grown mainly in the warmer Jordan Valley. There the disease is found in winter on these crops and on weeds.

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LONG SMUT OF SORGHUM IN ISRAEL[1]

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The local varieties of Sorghum vulgare generally referred to as durra are widely grown in the Middle East and known to be susceptible to both covered kernel smut, Sphacelotheca sorghi (Lk.) Clint., and head smut, S. reiliana (Kuehn) Clint. The former disease was first recorded in Israel by Reichert (5) and both smuts have been described by Minz (3). No other smut was encountered so long as only durra sorghum was grown, although the long smut is widespread in neighboring countries (1, 2).

The recently introduced Leoti and dwarf sorghum varieties are much less susceptible to the smuts mentioned above but were found to be hosts of the long smut, Tolyposporium ehrenbergii (Kuehn) Pat. (syn. T. filiferum Busse, Sorosporium ehrenbergii Kuehn.).

DESCRIPTION

The long smut of sorghum is characterized by long sori which are slightly or conspicuously curved (Fig. 1) and are scattered singly over infected heads (4), especially near their top. This feature distinguishes the long smut macroscopically from loose kernel smut, Sphacelotheca cruenta (Kuehn) Potter. Long smut sori measured 13 to 36 mm (mean 23.5 mm), ruptured generally at the apex, and split longitudinally exposing dark brown to black filaments surrounded by spore masses. Microscopically, long smut spores are found to be echinulate and to aggregate in masses (balls). On germination, a somewhat elongated promycelium is formed. However, ramification of this promycelium could not be followed because of contamination by bacteria which are always present in the spore balls.

FIGURE 1. Sori of long smut on a head of dwarf sorghum.

DISTRIBUTION IN ISRAEL

In the 5-year period 1955-1959 the smut was recorded during the months from July to September of four summers, in the Huleh Valley, especially in the southern part. Only in September 1955 was the disease also found in the Jordan Valley and at isolated points of the Coastal Plain. A survey in 1959 failed to reveal the smut anywhere in the Coastal Plain and Western Negev.

In the Huleh Valley, summer months are characterized by low relative humidities, with monthly mean values of 50 to 55 percent and daily minima of 35 to 40 percent for the months of July -August. This contrasts with monthly means of 70 to 80 percent and daily minima of 50 to 55 percent for the same months in the Coastal Plain. The common occurrence of T. ehrenbergii in the Huleh Valley would appear to indicate a preference for dry conditions.

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2 Plant Pathologists.

WORLD DISTRIBUTION

T. ehrenbergii has been recorded on sorghum only and on no other host. The world distribution of this smut, as gleaned mainly from the Review of Applied Mycology, is as follows:

1. Africa: Tanganyika (Rev. Appl. Mycol. 11: 235, 1932), Somaliland (Rev. Appl. Mycol. 16: 598, 1937), Sudan (Rev. Appl. Mycol. 36: 752, 1957), and Egypt where the smut was described by Briton-Jones (1) and by Melchers (2).

2. Asia: China (Rev. Appl. Mycol. 33: 450, 1954), Soviet Central Asia (Rev. Appl. Mycol. 5: 174, 1926), Iraq (Rev. Appl. Mycol. 32: 473, 1953), and several parts of India, such as Mysore (Rev. Appl. Mycol. 36: 518, 1957), Madras (Rev. Appl. Mycol. 33: 525, 1954), the Punjab (Rev. Appl. Mycol. 32: 493, 1953) and Sind (Rev. Appl. Mycol. 25: 32, 1946).

3. Europe: Italy ? (Rev. Appl. Mycol. 18: 517, 1939).

The long smut of sorghum has not been recorded in the Western Hemisphere, except where it was intercepted by the U. S. Quarantine Service on a consignment of sorghum from India.

This restriction to the Eastern Hemisphere seems to apply to all pathogenic species of the genus Tolyposporium. The most common species of this genus, T. penicillariae, affecting Pennisetum typhoides, occurs only in Asia and Africa (Commonwealth Mycological Institute Map No. 150). Other species of the genus are T. globuligerum reported from French Morocco (Rev. Appl. Mycol. 36: 555, 1957), T. andropogonis (Rev. Appl. Mycol. 32: 38, 1953) from India, T. restifaciens from New South Wales (Rev. Appl. Mycol. 32: 630, 1953), T. paspali from Queensland (Rev. Appl. Mycol. 29: 279, 1950), and T. volkensii from East Africa (Rev. Appl. Mycol. 18: 517, 1939).

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